CLINICAL AND EPIDEMIOLOGICAL STUDIES OF BOVINE NEOPLASIA

TWO VOLUMES

VOLUME 1

by

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TABLE OF CONTENTS

VOLUME 1

CHAPTER 1

A SURVEY OF MALIGNANT NEOPLASMS AND ASSOCIATED BENIGN NEOPLASMS OF CATTLE

CHAPTER 2

A CLINICO-PATHOLOGICAL STUDY OF UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA, URINARY BLADDER NEOPLASIA AND LYMPHOSARCOMA IN CATTLE

CHAPTER 3

AN EPIDEMIOLOGICAL STUDY OF BOVINE NEOPLASIA WITH PARTICULAR REFERENCE TO ALIMENTARY AND URINARY BLADDER NEOPLASMS AND THEIR RELATIONSHIP WITH BRACKEN FERN (PTERIDIUM AQUILINUM)

VOLUME 2

APPENDIX 1

APPENDIX 2

APPENDIX 3

REFERENCES

LIST OF FIGURES

LIST OF TABLES

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W.T.R. Grimshaw
November 30th, 1983

DECLARATION

I declare that the work presented in this thesis has been carried out by me. The pathology was done in conjunction with the members of the Department of Veterinary Pathology, in particular Mrs. P.E. McNeil, and the statistical analyses in conjunction with Mr. D. Hole, Cancer Epidemiology Unit, Ruchill Hospital, Glasgow.

Some of the material in this thesis has been published in the following papers :

(1) Jarrett, W.F.H., McNeil, P.E., Grimshaw, W.T.R., Selman, I.E. and McIntyre, W.I.M. (1978). High incidence area of cattle cancer with a possible interaction between an environmental carcinogen and a papilloma virus. Nature, 274, 215.

(2) Grimshaw, W.T.R. (1978). The epidemiology of various alimentary and urinary bladder neoplasms of cattle. Proceedings of the Academic Society for Large Animal Medicine, 1978, Berne.

(3) Grimshaw, W.T.R., Wiseman, A., Petrie, L. and Selman, I.E. (1979). Bovine leukosis (lymphosarcoma): A clinical study of 60 pathologically confirmed cases. Veterinary Record, 105, 267.

(4) Grimshaw, W.T.R., Wiseman, A., Petrie, L. and Selman, I.E. (1979). The major clinical and epidemiological features of bovine lymphosarcoma in Britain. The State Veterinary Journal, 34, 120.

(5) Grimshaw, W.T.R. and Evans, I.A. (1981). The inter-relationship between bovine neoplasia and bracken fern. In: Oncology Supplement: Scientific Foundations of Oncology. Edited by T. Symington and R.L. Carter. Wm. Heinemann Medical Books, London, 1981, p49.

SUMMARY

Between 1968 and 1971 a number of cattle routinely admitted to the Department of Veterinary Medicine were found to be affected by upper alimentary squamous cell carcinoma which is generally considered to be a rare bovine neoplasm. In some of these animals neoplasia of the urinary bladder was also present. The simultaneous occurrence of these neoplasms in cattle whose farms of origin were found to have an unusually localised distribution prompted the clinical and epidemiological investigations presented in this thesis.

In a survey of bovine neoplasia conducted over the period 1/9/71 - 31/8/79, upper alimentary squamous cell carcinoma, lymphosarcoma, transitional cell carcinoma of the urinary bladder and intestinal adenocarcinoma accounted for over 80 percent of the 275 malignancies identified. Two or more malignancies were found in 19 animals and in each of these cases upper alimentary squamous cell carcinoma and,or malignant urinary bladder neoplasia and,or intestinal adenocarcinoma were present. In addition, three types of benign neoplasia; upper alimentary papillomas, benign urinary bladder neoplasms and adenomas or adenomatous hyperplasia of the intestine, were frequently encountered at post mortem examination of animals with alimentary and urinary bladder malignancies.

Clinical examination of 55 animals with upper alimentary squamous cell carcinoma revealed four distinct clinical syndromes which could be correlated with the sites of carcinoma identified

at neocropsy; (1) an oropharyngeal syndrome characterised by halitosis, dribbling of saliva, coughing, snoring and the presence of an oropharyngeal mass, (2) an oesophageal syndrome characterised by cud-dropping, the presence of a mass in the cervical oesophagus, halitosis and diarrhoea, (3) a ruminal tympany syndrome characterised by ruminal tympany and profuse diarrhoea and (4) a wasting and diarrhoea syndrome characterised by poor body condition and profuse diarrhoea. Clinical examination of 27 animals with urinary bladder neoplasms revealed a syndrome characterised by haematuria. Other major clinical signs included pallor of the mucosae, due to anaemia, and abnormalities of the urinary tract which could be detected per rectum. Clinical examination of 64 animals with lymphosarcoma revealed three distinct clinical forms of the disease which correlated with the pathological distribution of the neoplasm; (1) a multicentric form characterised by generalised lymph node enlargement in immatures and localised lymph node enlargement in adults, (2) a thymic form characterised by the presence of a cervical and, or anterior thoracic mass, with the resultant clinical effects of obstruction of the thoracic inlet by this mass and (3) a skin form characterised by neoplastic infiltration of the skin and generalised lymph node enlargement.

The epidemiological aspects of bovine neoplasia with particular reference to bracken fern are examined in the final chapter. The prevalences of most types of neoplasms were found to increase with age but in the case of lymphosarcoma the opposite applied. Examination of the breed distribution of cattle affected

by neoplasia demonstrated that there was a highly significant association between upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia and beef breeds of cattle.

The geographical distribution of these alimentary and urinary bladder neoplasms was found to be localised in specific areas of Scotland whereas other neoplasms had a pattern of distribution similar to that of all cattle admitted to the Department of Veterinary Medicine during the period of the study. In addition, it was demonstrated that there was a highly significant association between the occurrence of these neoplasms and the severity of infestation by bracken fern in the referral areas of affected animals. This association was investigated in detail by means of a case controlled study. Highly significant associations were found between upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia and the presence of bracken fern, the severity of bracken infestation and the occurrence of acute bracken poisoning on the referral farms of affected animals. In addition, fifteen farms were identified from which between two and eight adult cattle with alimentary or urinary bladder malignancies had been referred, and it was shown that, on these farms bracken infestation was more severe and acute bracken poisoning incidents were more frequently recognised than on single case farms.

Although exposure to bracken fern appeared to be a pre-requisite for the development of upper alimentary

squamous cell carcinoma and urinary bladder neoplasms, a small number of animals with upper alimentary papillomas were identified which had apparently never had access to bracken. A study of the prevalence of palatine papillomas on bracken infested and bracken free farms confirmed that there was usually a high prevalence of palatine papillomas in cattle aged over 15 months which had been exposed to bracken fern, but that their prevalence on bracken free farms was extremely low. The aetiological implications of these findings with respect of alimentary and urinary bladder neoplasia in cattle are discussed in light of present knowledge regarding the carcinogenicity of bracken.

GENERAL INTRODUCTION

Detailed epidemiological investigations of bovine neoplasia have seldom been undertaken due to the numerous difficulties which are encountered in comparison with similar studies in man. Tjalma (1963) outlined some of the most important factors which place serious limitations on research of the epidemiology of animal neoplasms, which include the lack of basic data, because there is no requirement to register information such as birth dates and causes of death and there are no detailed population statistics. With particular reference to cattle other factors include the expense of obtaining affected animals because of their commercial value, and the often insurmountable problem of tracing their farm or farms of origin.

Epidemiological studies of bovine neoplasia have, with a few notable exceptions, been confined to the delineation of the prevalence or frequency of individual tumours and their age, breed and sex distribution. This data has mainly been obtained through surveys performed in abattoirs or at veterinary schools, and often has not only been based on examination of animals slaughtered or dying in these institutions but also on material submitted from elsewhere for diagnosis.

However, to date, large scale surveys have seldom led to further studies to identify variables of aetiological significance. The most productive investigations have

usually resulted from the identification of problems and features associated with a specific neoplasm such as a high prevalence of clinical disease e.g. urinary bladder neoplasms and ocular squamous cell carcinoma,or clinical disease combined with apparent farm to farm spread and high condemnation rates in local abattoirs e.g. lymphosarcoma.

During the late 1960s a similar situation became apparent in the routine examination of cattle referred to Glasgow University Veterinary Hospital. For many years, diseased cattle have been purchased by the Department of Veterinary Medicine for the purposes of undergraduate clinical teaching. These animals are obtained from farms throughout Scotland and the North of England by referral through practicing veterinary surgeons or, occasionally, from cattle dealers and markets.

Between 1968 and 1971 a number of animals affected by upper alimentary squamous cell carcinoma, a comparatively rare bovine neoplasm, were referred from the county of Argyll which is situated in western Scotland, north of Glasgow. Some of these animals were also found to be affected by urinary bladder neoplasia.

The simultaneous occurrence of neoplasia of two different sites, in cattle whose farms of origin had an unusually localised distribution, prompted the clinical and epidemiological study which forms the basis of this thesis.

For ease of presentation, the thesis has been divided into three chapters each of which is accompanied by a review of the relevant literature:

(1) A survey of the malignancies and associated benign neoplasms in cattle admitted to the Department of Veterinary Medicine, Glasgow University Veterinary Hospital during the eight year period, 1/9/71 - 31/8/79.

(2) A clinico-pathological study of the major groups of neoplasms identified in the survey, and

(3) An epidemiological study of the neoplasms identified in the survey.

CHAPTER 1

A SURVEY OF MALIGNANT NEOPLASMS AND ASSOCIATED BENIGN NEOPLASMS OF CATTLE

REVIEW OF THE LITERATURE

Although the information contained in many of the surveys of bovine neoplasia is very limited they provide a general indication of the relative frequencies of individual bovine neoplasms in various regions of the world. For example, lymphosarcoma is the malignancy most frequently recorded in surveys performed in the United Kingdom (Cotchin, 1960; Anderson, Sandison and Jarrett, 1969), the Netherlands (Misdorp, 1967), Czechoslovakia (Vitovec, 1976), New Zealand (Shortridge and Cordes, 1971) and Canada (Plummer, 1956) whereas in the United States of America, the frequency of ocular squamous cell carcinoma is much greater than that of lymphosarcoma (Monlux, Anderson and Davis, 1956; Brandly and Migaki, 1963). Similarly, Nair and Sastry (1953) state that in India ocular squamous cell carcinoma is the most commonly recognised malignancy of cattle, but they also report a high frequency of horn core carcinoma which is rarely recorded in western Europe and the United States. Other malignancies which have been shown to be relatively common in certain areas include adenocarcinoma of the uterus (Monlux and others, 1956; Brandly and Migaki, 1963; Vitovec, 1976), cholangiosarcoma (Anderson and others, 1969), hepatocellular carcinoma (Vitovec, 1976), lung carcinoma (Brandly and Migaki, 1963; Anderson and others, 1969), squamous cell carcinoma of the vulva and perineum (Shortridge and Cordes, 1971) and intestinal adenocarcinoma (Misdorp, 1967;

Vitovec, 1976).

Attempts have been made by some authors to define the incidence of different neoplasms (Brandly and Migaki, 1956; Anderson and others, 1969) but the accuracy of abattoir surveys in the assessment of the relative or true incidence must be suspect in many cases. For example, animals affected by tumours which only involve a localised site,e.g. ocular squamous cell carcinoma,may usually be sent to abattoirs, whereas those affected by malignancies which tend to have a widespread distribution throughout the body, e.g. lymphosarcoma, are much less likely to be disposed of in this way. In addition, abbatoir surveys take no account of animals which have died on farms,or immature animals which are not sent to abattoirs because they have little or no commercial value as a source of meat.

From the literature it is apparent that, even in countries where agriculture is highly developed, there is no concise data on the incidence of bovine neoplasia. In addition, there are only general indications of the relative frequencies of individual neoplasms, based on surveys which, in most cases, are comparatively small in numbers of animals examined, and localised in terms of the catchment area from which the animals originate.

A SURVEY OF MALIGNANT NEOPLASMS AND ASSOCIATED BENIGN NEOPLASMS OF CATTLE

INTRODUCTION

Despite the shortcomings of surveys of bovine neoplasia, the data collected can provide a basis for further investigations directed at the identification of factors which could account for any variations in the frequency of specific neoplasms when comparisons are made with previous surveys.

The following survey is presented as a basis for the detailed clinical and epidemiological studies which follow in Chapters 2 and 3 of this thesis.

MATERIALS AND METHODS

(1) Animals

The animals were referred to the Department of Medicine of Glasgow University Veterinary Hospital by practising veterinary surgeons in Scotland and the north of England and, to a lesser extent, by cattle dealers operating in the west of Scotland. The majority of these referrals were made for two reasons; their suitability as material for undergraduate clinical teaching or the need for a definitive diagnosis. In this study, only those animals which were subsequently submitted for post mortem examination are considered.

(2) Post Mortem Examinations

After slaughter, appropriate tissues were taken and fixed within two hours of death or, in those animals which died naturally, within twelve hours. Thereafter in all suspected cases of neoplasia, the gross pathological diagnosis was confirmed by standard histopathological techniques.

RESULTS

(1) Malignant Neoplasms

During the eight year period, 1/9/71 to 31/8/79, 2809 cattle were admitted to the Medicine Department of Glasgow University Veterinary Hospital and subsequently submitted for post mortem examination. Post mortem

examinations revealed that 254 animals (9.0%) were affected by malignant neoplasms and, of these, 19 were found to have two or more different malignancies. In total, 275 malignancies were identified. Four sites accounted for over 80 percent of the malignancies; the upper alimentary tract (35%), the lymphoid organs (28%), the urinary bladder (12%) and the intestines (7%)(Table 1). Squamous cell carcinoma was the only malignancy identified in the upper alimentary tract but was found in many sites including the tongue, palate, pharynx, oesophagus, cardia and anterodorsal sac of the rumen (Figures 1-6). In addition, in individual animals, there were frequently multiple primary foci of squamous cell carcinoma involving two or more of these sites (Figures 7 and 8). Lymphosarcoma was the sole primary malignancy of the lymphoid organs which was recognised (Figure 9). In 44 animals lymphosarcoma appeared to have arisen in the lymph nodes whereas in the remaining 33 animals the primary site was the thymus. Several different urinary bladder malignancies were recognised (Table 1) but transitional cell carcinoma was, by far, the most common (Figure 10). In the intestines, adenocarcinoma accounted for all but one of the malignancies identified and, with one exception were situated in the small intestine (Figure 11). The animals with multiple malignancies were all found to be affected by upper alimentary squamous cell carcinoma and, or malignant urinary bladder neoplasia and, or adenocarcinoma (Table 2). Lymphosarcoma was never identified in any of the animals with multiple malignancies. The types and sites of malignant neoplasms identified in the survey are recorded in Appendix 1.

TABLE 1

The Site and Type of Malignant Neoplasms

Identified between 1/9/71 and 31/8/79

Site and Type of Malignancy	Number
Upper Alimentary Tract	
Squamous cell carcinoma of the buccal cavity, pharynx, oesophagus and rumen	97
Secondary Lymphoid Organs	
Multicentric lymphosarcoma	44
Thymic lymphosarcoma	33
Urinary Bladder	
Transitional cell carcinoma	24
Haemangiosarcoma	5
Adenocarcinoma	2
Squamous cell carcinoma	1
Intestines	
Adenocarcinoma	18
Other Sites	
Various	51
Total	275

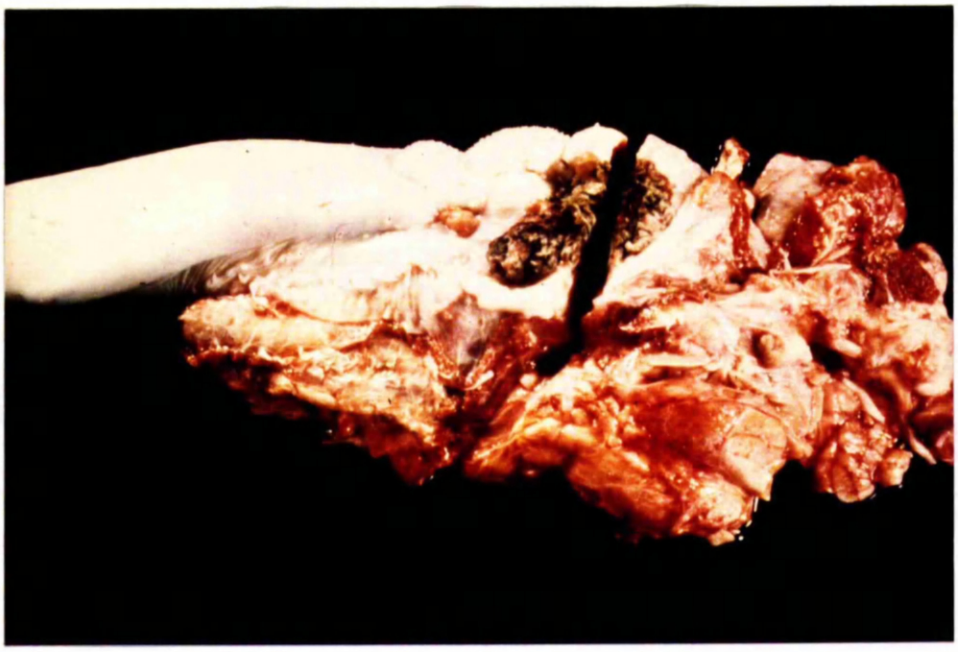


FIGURE 1 Squamous cell carcinoma of the tongue

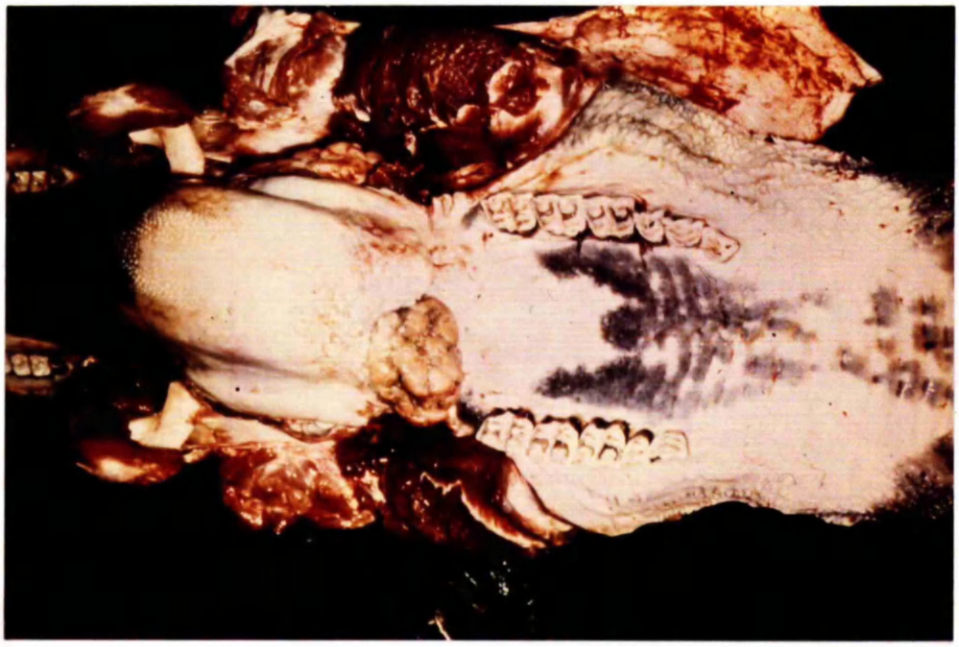


FIGURE 2 Squamous cell carcinoma of the palate

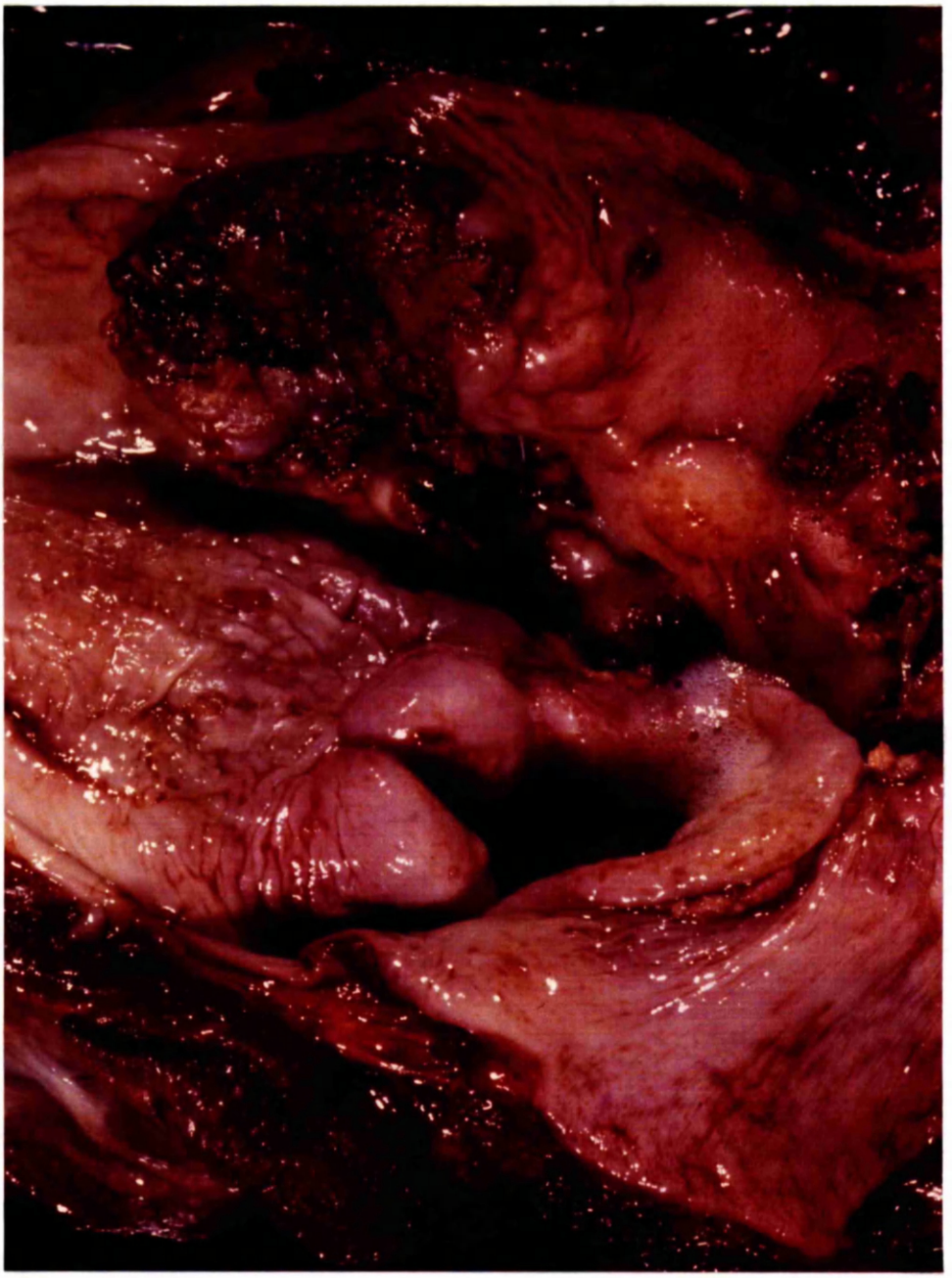


FIGURE 3 Squamous cell carcinoma of the pharynx

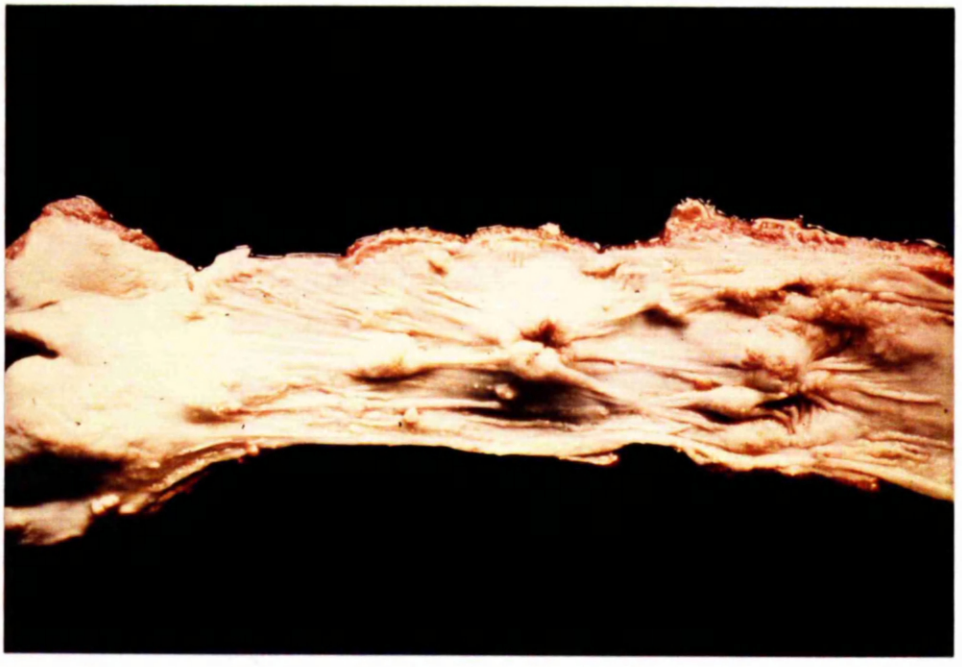


FIGURE 4 Squamous cell carcinoma of the oesophagus

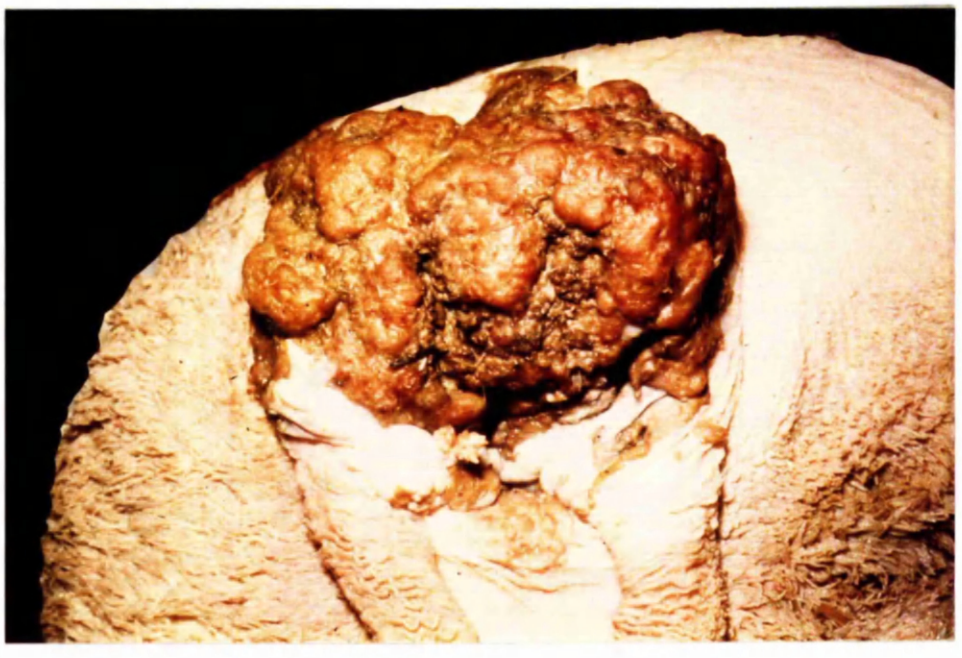


FIGURE 5 Squamous cell carcinoma of the cardia.

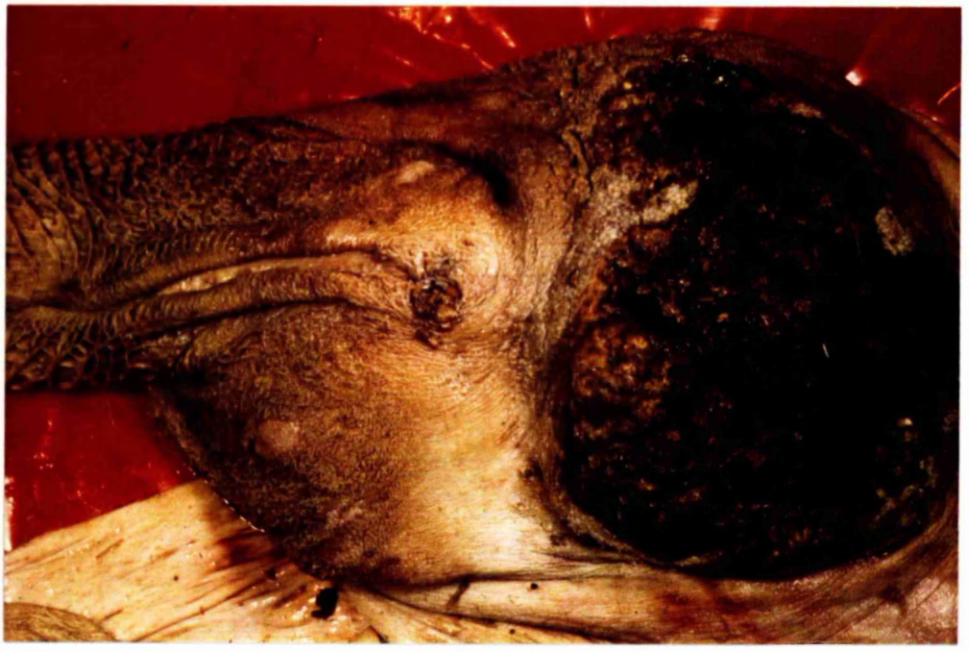


FIGURE 6 Squamous cell carcinoma of the rumen and cardia

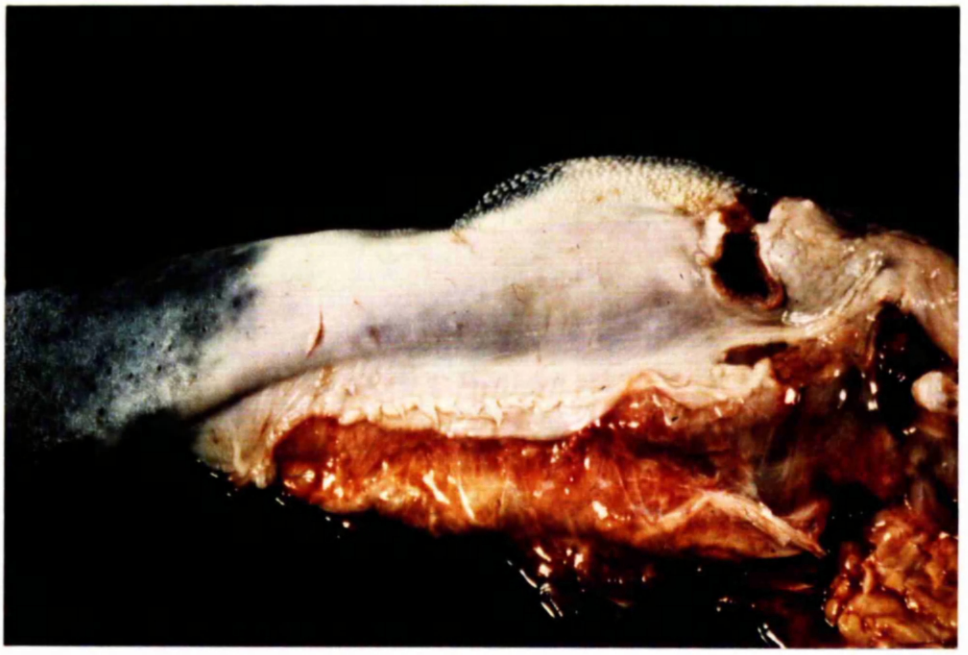


FIGURE 7 Squamous cell carcinoma of the tongue
(Same animal as Figure 8)

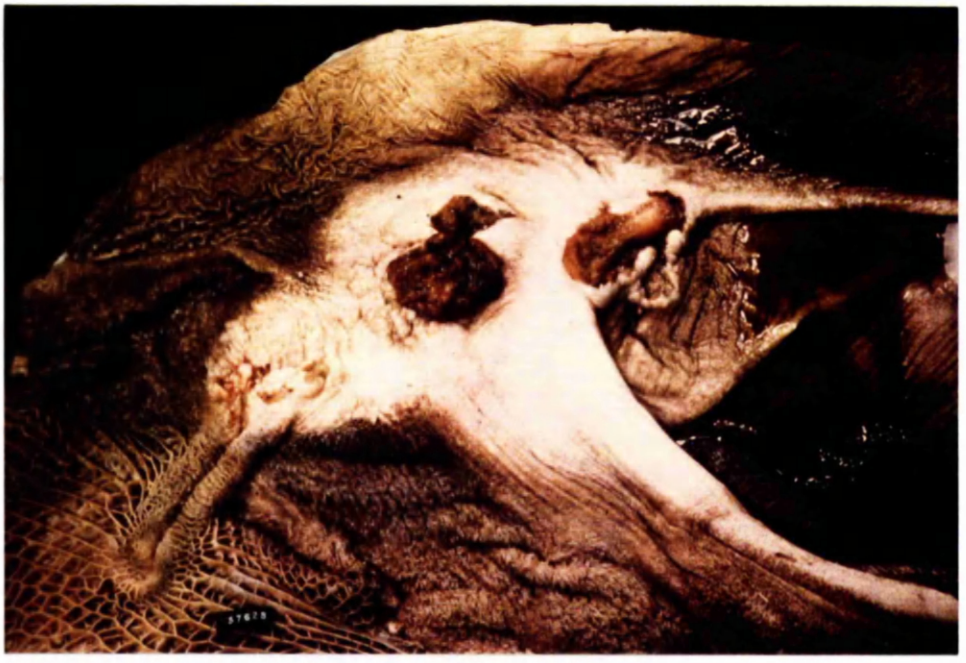


FIGURE 8 Squamous cell carcinoma of the cardia
and rumen

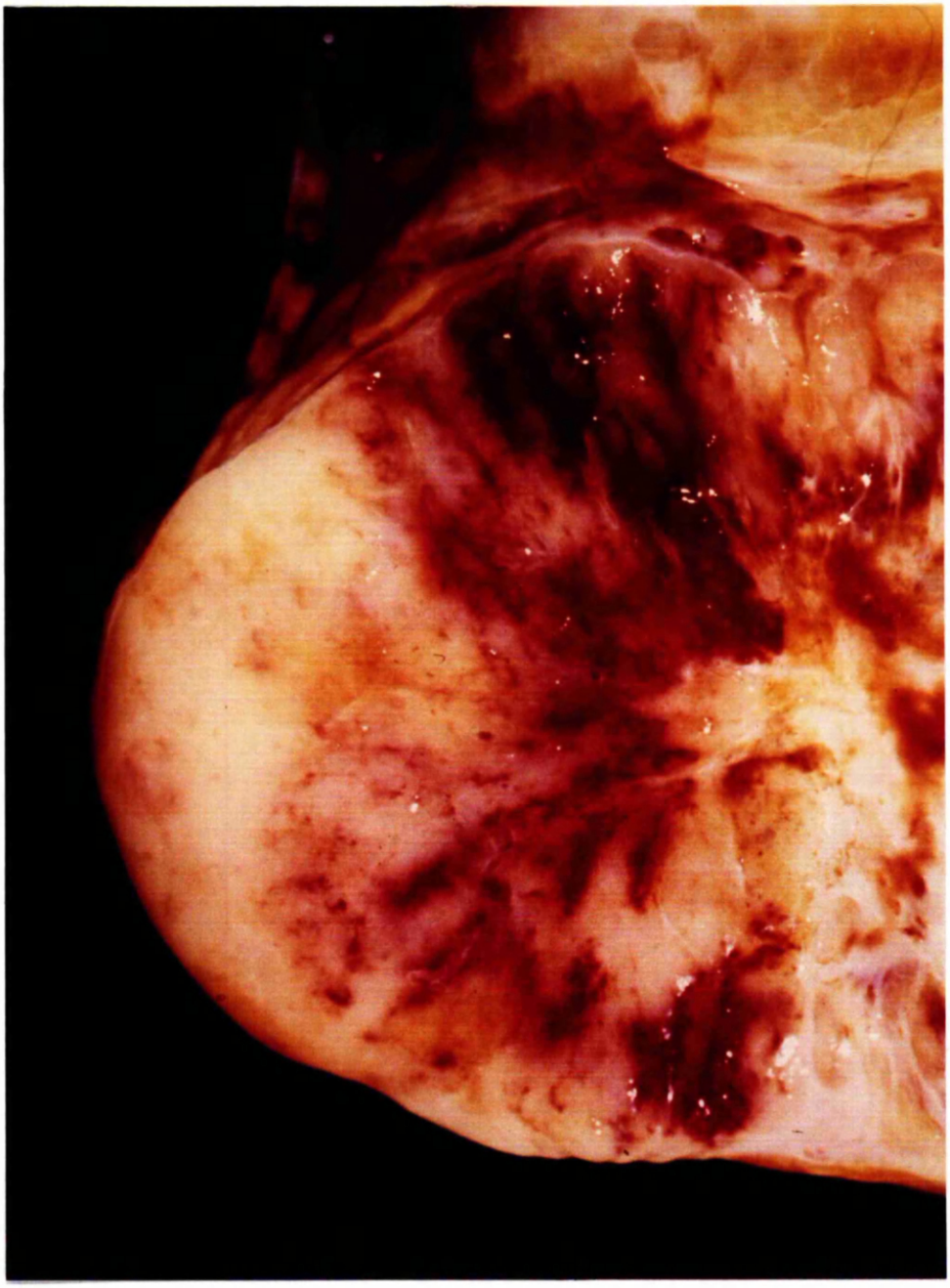


FIGURE 9 Multicentric lymphosarcoma.
Section of a superficial cervical lymph node

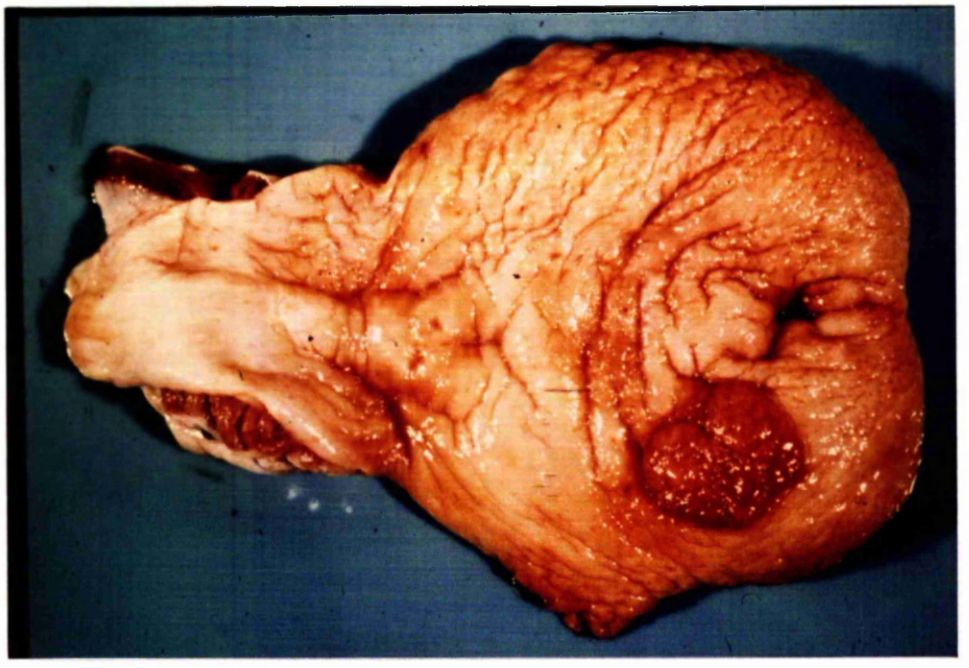


FIGURE 10 Transitional cell carcinoma of the urinary bladder

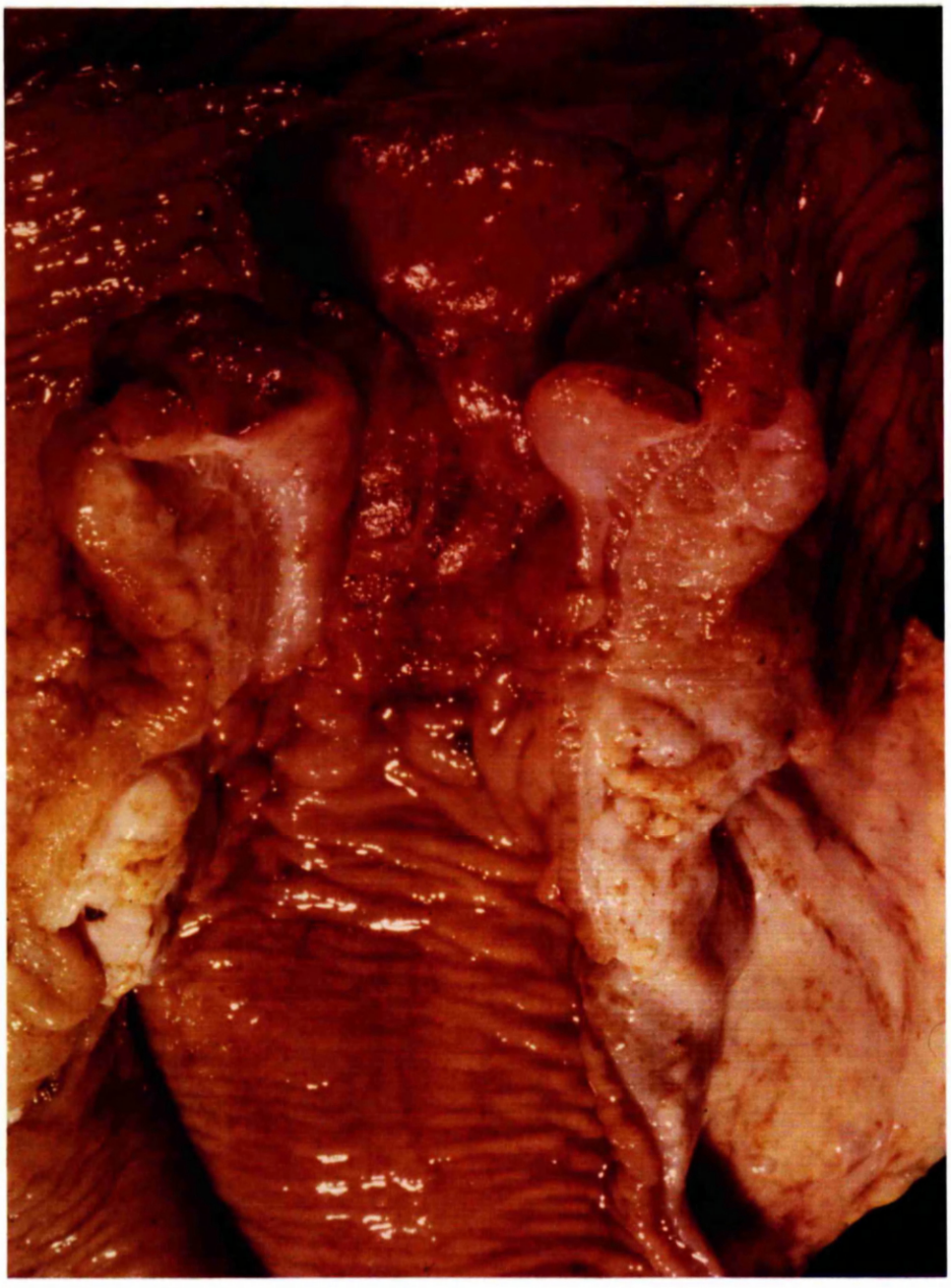


FIGURE 11 Intestinal adenocarcinoma

TABLE 2

The Malignant Neoplasms Present in Animals with Multiple Malignancies

Neoplasms	Neoplasms Identified						
Upper Alimentary Squamous Cell Carcinoma	*	*	*	*	*		17
Urinary Bladder Transitional Cell Carcinoma	*	*				*	8
Urinary Bladder Haemangiosarcoma			*				2
Urinary Bladder Adenocarcinoma		*					1
Intestinal Adenocarcinoma				*	*	*	11
Squamous Cell Carcinoma of the Vagina					*		1
Number of Animals	5	1	2	8	1	2	

Total Number of Animals = 19

Total Number of Malignancies = 40

(2) Benign Neoplasms Associated with Malignant Neoplasia

Three forms of benign neoplasia were frequently encountered at post-mortem examination of cattle affected by malignancies; squamous papillomas of the upper alimentary tract, benign urinary bladder neoplasms and intestinal adenomas and, or adenomatous hyperplasia (Figures 12 - 14).

Upper alimentary papillomas were present in 125 (49%) of the cattle with malignancies (Table 3). They were particularly common in animals affected by upper alimentary squamous cell carcinoma, malignant urinary bladder neoplasia and intestinal adenocarcinoma of which 97 per cent, 77 per cent and 78 per cent respectively were found to have upper alimentary papillomas. None of the animals with lymphoscarcoma had upper alimentary papillomas but they were found in 25 per cent of the animals with other malignant neoplasms. The numbers of upper alimentary papillomas present in animals with malignancies was extremely variable and ranged between one and over 75 but the distribution of papillomas was identical to that of upper alimentary squamous cell carcinoma. The details of the relationship between upper alimentary papillomas and malignancy will be examined at a later stage.

Benign urinary bladder neoplasms were present in 24 (9%) of the animals with malignancies (Table 4) but were mainly confined to those affected by malignant urinary bladder neoplasia or upper alimentary squamous cell carcinoma of which 42 per cent and 12 per cent had benign urinary bladder neoplasia respectively. Four (22%) of the cattle affected by intestinal adenocarcinoma were found to have

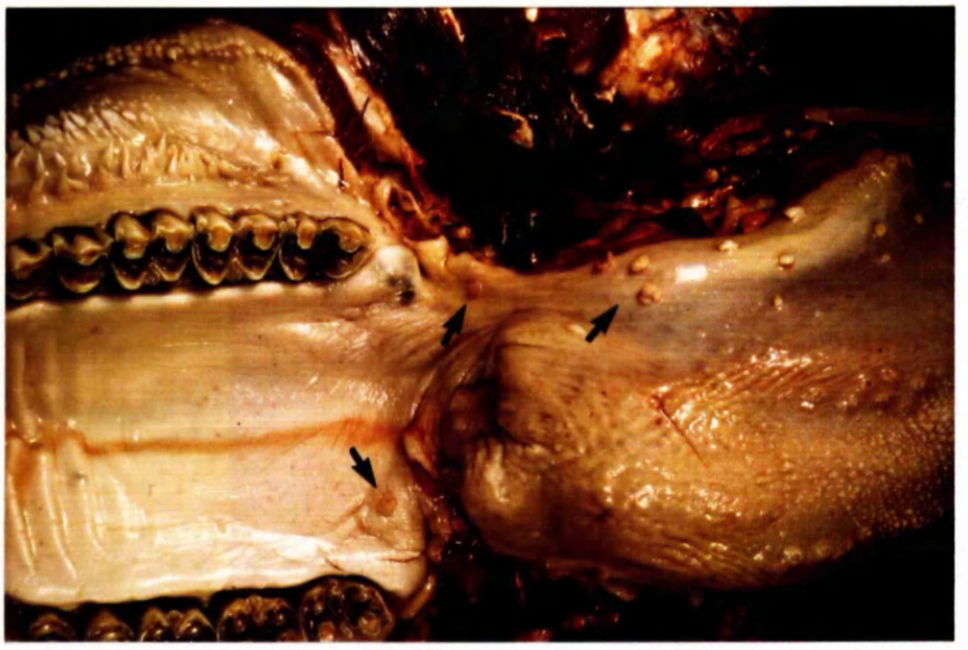


FIGURE 12 Squamous papillomas of the palate and tongue

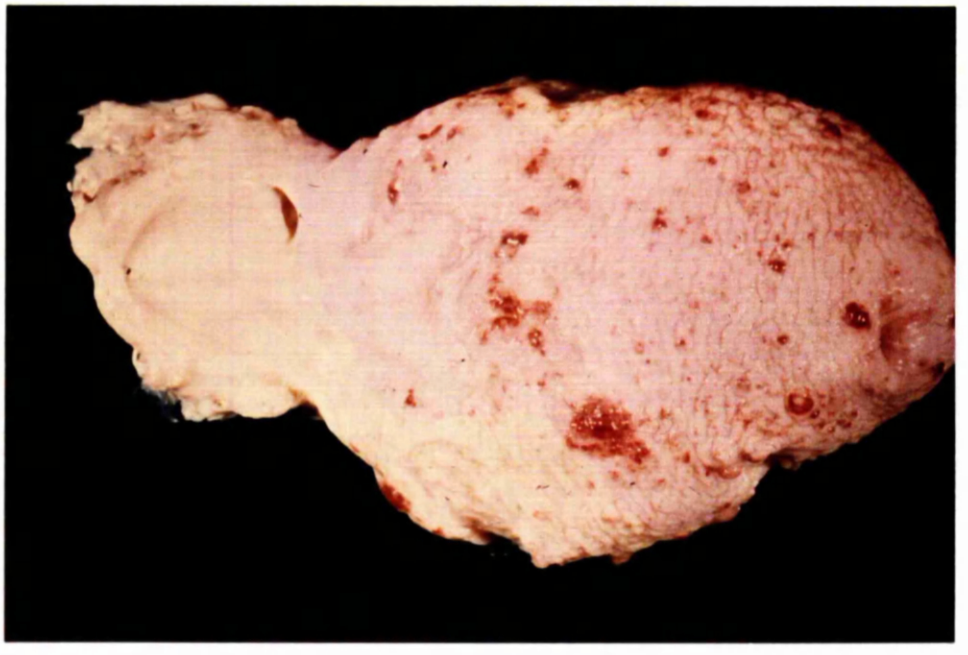


FIGURE 13 Haemangiomas of the urinary bladder

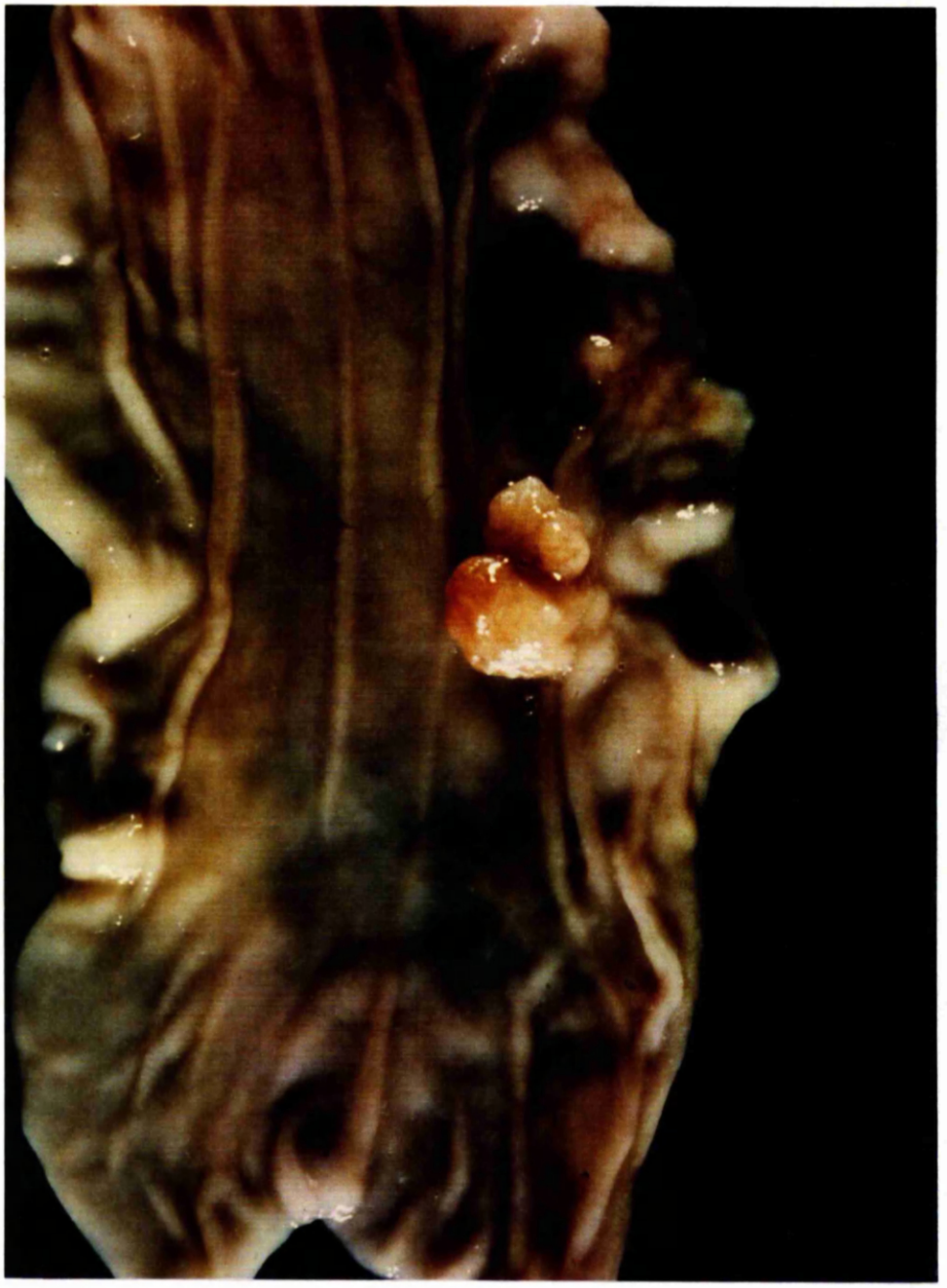


FIGURE 14 Intestinal adenoma

TABLE 3

The Presence of Upper Alimentary Papillomas in Animals with Malignancies

Malignancy	Number of Animals *	Number with Papillomas *	Percentage with Papillomas *
Upper Alimentary Squamous Cell Carcinoma	97 (17)	94 (17)	97 (100)
Malignant Urinary Bladder Neoplasms	31 (10)	24 (9)	77 (90)
Intestinal Adenocarcinoma	18 (11)	14 (10)	78 (91)
Lymphosarcoma	77 (0)	0 (0)	0 (0)
Other Malignant Neoplasms	51 (1)	13 (1)	25 (100)
All Animals with Malignancies	254 (19)	125 (18)	49 (95)

* The values for animals with multiple malignancies are given in brackets.

TABLE 4

The Presence of Benign Urinary Bladder Neoplasms

in Animals with Malignancies

Malignancy	Number of Animals *	Number with Benign Urinary Bladder Neoplasms *	Percentage with Benign Urinary Bladder Neoplasms *
Upper Alimentary Squamous Cell Carcinoma	97 (17)	12 (4)	12 (24)
Malignant Urinary Bladder Neoplasms	31 (10)	13 (3)	42 (30)
Intestinal Adenocarcinoma	18 (11)	4 (3)	22 (27)
Lymphosarcoma	77 (0)	0 (0)	0 (0)
Other Malignant Neoplasms	51 (1)	0 (0)	0 (0)
All Animals with Malignancies	254 (19)	24 (5)	9 (26)

* The values for animals with multiple malignancies are given in brackets

benign urinary bladder neoplasms but only one of these did not have multiple malignancies. Two types of benign urinary bladder neoplasms were identified; haemangiomas which were present in 22 animals and fibromas which were present in three animals, there being one animal with multiple malignancies in which both types were found. There was no evidence of benign urinary bladder neoplasia in any of the cattle with lymphosarcoma or other malignant neoplasms.

Intestinal adenomas and, or adenomatous hyperplasia were present in 64 (25%) of the animals with malignancies (Table 5). They were most frequently found in small numbers in the duodenum and jejunum but in a few animals over 100 individual lesions were present, scattered throughout the small and large intestines. Amongst the animals with malignancies, intestinal adenomas and, or adenomatous hyperplasia were mainly confined to those affected by upper alimentary squamous cell carcinoma, malignant urinary bladder neoplasia and intestinal adenocarcinoma of which 53 per cent, 55 per cent and 39 per cent were affected respectively. They were not identified in any of the animals affected by lymphosarcoma and only in a very small proportion (4%) of those with other malignant neoplasms.

In addition to the 125 animals with malignant neoplasms which were found to have upper alimentary papillomas, a further 109 animals admitted during the eight year period had upper alimentary papillomas in the absence of any malignancy, the numbers of papillomas ranging

TABLE 5

The Presence of Intestinal Adenomas and/or Adenomatus Hyperplasia in Animals with Malignancies

	Number of Animals	Number with Adenomas and,or Adenomatous Hyperplasia	Percentage with Adenomas and,or Adenomatous Hyperplasia
Upper Alimentary Squamous Cell Carcinoma	97 (17)	51 (12)	53 (71)
Malignant Urinary Bladder Neoplasms	31 (10)	17 (8)	55 (80)
Intestinal Adenocarcinoma	18 (11)	7 (6)	39 (55)
Lymphosarcoma	77 (0)	0 (0)	0 (0)
Other Malignant Neoplasms	51 (1)	2 (0)	4 (0)
All Animals with Malignancies	254 (19)	64 (13)	25 (68)

between one and over 25. Similarly, in addition to the 24 animals with malignant neoplasms which were found to have benign urinary bladder neoplasms, a further 17 animals had benign urinary bladder neoplasms in the absence of any malignancy. Thirteen of these animals had haemangiomas, three had fibromas and one was affected by both neoplasms simultaneously. Twelve (71%) of these animals with benign urinary bladder neoplasia also had upper alimentary papillomas.

Adenomas and, or adenomatous hyperplasia were identified in 24 per cent and 10 per cent respectively of animals with benign urinary bladder neoplasms or upper alimentary papillomas in the absence of any malignancy. However these lesions were only rarely recorded in other animals unaffected by malignancy which were admitted during the period of study. It is probable that this is a reflection of the lack of detailed examination of the entire intestinal tract, which in adult cattle measures between 33 and 63 metres, and thus the failure to identify these relatively insignificant lesions.

Thirty other benign neoplasms were found in animals affected by malignant neoplasia (Appendix 1). Small, non invasive phaeochromocytomas which were considered to be benign were identified in four animals with upper alimentary squamous cell carcinoma. In addition, fibroma of the oesophagus or rumen, adenoma of the gall bladder, lipoma of the colon and fibropapilloma of the teats were each present in two to four animals, but all the remaining types of benign neoplasm were only identified on one occasion.

DISCUSSION

The initial stimulus for this survey of bovine neoplasms was the identification of a number of examples of an apparently rare neoplasm, upper alimentary squamous cell carcinoma, in cattle and pathological specimens submitted to Glasgow University Veterinary Hospital for diagnosis. Six of these cases have been described by Pirie (1973). The results of the study performed on cattle admitted to the Medicine Department of the Veterinary Hospital between 1971 and 1979 not only demonstrate that, within the catchment area of admissions, a high frequency of upper alimentary squamous cell carcinoma exists relative to other malignancies, but that there is also a high frequency of other neoplasms which have seldom been recorded in previous surveys of bovine neoplasia.

In the present study, upper alimentary squamous cell carcinoma was the most frequently observed malignant neoplasm, followed by lymphosarcoma, transitional cell carcinoma of the urinary bladder and intestinal adenocarcinoma. However, the results of most other studies performed in the United Kingdom, including the major countrywide abattoir survey of Anderson and others (1969), have shown that lymphosarcoma is the pre-iminent malignant neoplasm of cattle, and would tend to suggest that, in comparison, the other three neoplasms are either uncommon or rare. The comparative rarity of upper alimentary squamous cell carcinoma and all forms of malignant urinary bladder neoplasia is also evident from surveys

of bovine neoplasia conducted elsewhere in the world, irrespective of which neoplasms are the most frequently observed. Similarly, although Misdorp (1967) stated that, in his study, the most common tumour of the alimentary tract was adenocarcinoma of the jejunum, intestinal adenocarcinoma is infrequently recognised in cattle, as is apparent from the extensive review of the literature performed by Lingeman and Garner (1972) who were able to find only 36 recorded cases.

However, there are reports which indicate that both upper alimentary squamous cell carcinoma and malignant urinary bladder neoplasia can occur with high frequency in cattle populations. Over 70 years ago, Trotter (1911) found that upper alimentary squamous cell carcinoma was the most common malignancy of cattle examined in an abattoir study carried out in Glasgow, Scotland, although based mainly on animals imported from Ireland for slaughter. Other reports of a high frequency of either upper alimentary squamous cell carcinoma or malignant urinary bladder neoplasia have seldom been the result of surveys of bovine neoplasia but have arisen due to the recognition of clinical disease within a localised geographical area. A detailed appraisal of the significance of these reports to this study is made at a later stage.

The presence of two or more different types of malignancies in individual cattle, as observed in 19 of the animals examined in this study, has seldom been reported. In addition, it would appear to be significant that in all these animals two of the following were present; upper

alimentary squamous cell carcinoma, malignant urinary bladder neoplasia and intestinal adenocarcinoma. The occurrence of a comparatively high frequency of each of these neoplasms combined with their simultaneous presence in individual aminals could indicate that either a common aetiological factor(s) is responsible, or that several mutually exclusive factors are operating in the same time and place. Similarly, the high frequency with which upper alimentary papillomas and benign urinary bladder neoplasms were found in animals affected by alimentary and urinary bladder malignancies suggests that these benign neoplasms could also be related to the same factor(s).

Although adenomas and adenomatous hyperplasia of the intestine were only identified in animals with malignancies, the lack of information regarding their prevalence in other animals, unaffected by malignant neoplasia, prevents any interpretation of their significance and precludes any assessment of their relationship to malignancy in later parts of this study.

The only other benign neoplasms which were found in more than two animals with malignancies were fibroma of the oesophagus and rumen, fibropapilloma of the teats and phaeochromocytoma of the adrenal gland. Upper alimentary fibromas and teat fibropapillomas are generally considered to be common bovine neoplasms. Similarly, phaeochromocytoma of the adrenal gland is well recognised in cattle (West, 1975;

Appleby, 1976) and although it has not been previously found in animals with upper alimentary squamous cell carcinoma, it has been found simultaneously with ultimobranchial tumours in bulls (Wilkes and Krook, 1970) and a thyroid carcinoma in a cow (Charan, Gill and Parihar, 1976).

CHAPTER 2

A CLINICO-PATHOLOGICAL STUDY OF UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA, URINARY BLADDER NEOPLASIA AND LYMPHOSARCOMA IN CATTLE

REVIEW OF THE LITERATURE

Upper Alimentary Squamous Cell Carcinoma

There are extremely few descriptions of the clinical syndromes associated with squamous cell carcinoma of the upper alimentary tract, and diagnosis of the condition on a clinical basis has been confined to the localised areas of Brazil and Kenya in which a high prevalence of the neoplasm is recognised.

In Brazil, Dobereiner, Tokarnia and Canella (1967), Tokarnia, Dobereiner and Canella (1969) and Campos Neto, Barros and Bicudo (1975) recognised two clinical syndromes associated with the neoplasm. The first was characterised by weight loss, coughing, snoring, and various manifestations of difficulty in swallowing and eating, including drooling of saliva and regurgitation of food through the nares and from the mouth. An oropharyngeal mass or large ulcer was frequently detected clinically and in some animals, diarrhoea, halitosis and oropharyngeal papillomata were seen. This syndrome was associated with the presence of squamous cell carcinoma of the oropharynx, or, less commonly, of the proximal oesophagus.

The second syndrome was characterised by ruminal tympany or, regurigtation and loss of cud from the mouth. Weight loss and diarrhoea were frequently present and, in some cases, there was difficulty in passing a stomach tube due to an obstruction in the oesophagus. This syndrome

was associated with the presence of a squamous cell carcinoma in the distal oesophagus or at the cardia.

Similarly, in Kenya, Plowright (1955) described a syndrome of chronic ruminal tympany and loss of weight in animals which were found, on post mortem examination, to have squamous cell carcinoma affecting the rumen in the area of the oesophageal groove or the distal oesophagus. The initial, very brief, clinical description was augmented by Plowright, Linsell & Peers (1971) who stated that, in addition to recurrent ruminal tympany, the clinical signs which characterised the disease were; apparent pain or difficulty in swallowing,or regurgitation of food during rumination including, in some animals, regurgitation of watery rumen contents from the mouth and through the nostrils, abdominal pain, loss of condition and, occasionally, partial anorexia, slow eating and excessive thirst.

A small number of clinical cases have also been described by workers in Scotland. Rumenal tympany was the main clinical feature in a single case described by Wood, Jennings and McIntyre (1957) in which a large ulcerated carcinoma was situated in the ruminal wall. Pirie (1973) described six cases in which difficulty in swallowing was associated with a palpable oesophageal mass or obstruction to passage of a stomach tube,due to the presence of squamous cell carcinoma of the oesophagus.

Pathological investigations by a number of authors have revealed that squamous cell carcinoma of the upper alimentary tract can affect the tongue, hard and soft palates,

pharynx, oesophagus and rumen.

In addition, in individual animals, foci of carcinoma can be found simultaneously in more than one of these sites (Plowright, 1955; Dobereiner and others, 1967; Tokarnia and others, 1969; Plowright and others, 1971; Pirie, 1973) and it has been suggested that this is due to multicentric origin of the carcinoma rather than metastatic spread.

The rumen is the site in which the carcinoma has been recorded most frequently and in this organ the carcinoma is usually situated in the anterior dorsal sac (Trotter, 1903; Tokarnia and others, 1969; Plowright and others, 1971) where it assumes one of two gross morphological forms. The carcinoma may be either flattened, ulcerated and schirrous (Trotter, 1903; Nieberle and Cohrs, 1949; Plowright, 1955; Wood and others, 1957; Plowright and others, 1971) or broad based, projecting and cauliflower-like in appearance (Trotter, 1903; Vlalukin, 1928; Nieberle and Cohrs, 1949; Damodaran, 1959; Tokarnia and others, 1969; Plowright and others, 1971). Frequently these carcinomas are extremely large in size. Plowright (1955) Wood and others (1957) record examples of the flattened ulcerated form in excess of 10 cms in diameter and in one case described by Trotter (1903) the dimensions were 50 x 18 cms. Similarly, large examples of the cauliflower-like, projecting form have been described ranging from 10 cms to in excess of 23 cms in diameter (Trotter, 1903; De Kock and Fourie, 1928; Damodaran, 1959; Tokarnia and others, 1969).

In the oesophagus, squamous cell carcinoma is usually a stenosing, ulcerated lesion (Dobereiner and others, 1967; Tokarnia and others, 1969; Pirie, 1973), and can occur at any site along the length of the oesophagus including the cervical portion (Tokarnia and others, 1969; Pirie, 1973), the distal intrathoracic portion (Plowright, 1955) and at the cardia (Dobereiner and others, 1967).

Squamous cell carcinomas of the tongue, palate and pharynx are usually eroding and ulcerative (Dobereiner and others, 1967; Tokarnia and others, 1969; Pirie, 1973) but can be pedunculated and nodular in appearance (Tokarnia and others, 1969). When the tongue is involved the lesion tends to be confined to the base of the organ where it can infiltrate deeply into the surrounding tissues (Tokarnia and others, 1969; Pirie, 1973).

Metastatic spread of upper alimentary squamous cell carcinoma is relatively uncommon and is usually confined to the local lymphatic drainage of the site involved. However,metastases from a primary site in the oesophagus and rumen have been observed in the following organs; the atrial and posterior mediastinal lymph nodes, the omentum, the visceral and parietal peritoneum, the liver and hepatic lymph nodes, the lumbar lymph nodes, the kidneys and renal lymph nodes,and the lungs (Trotter, 1903, 1911; Tokarnia and others, 1969; Plowright and others, 1971). Metastases from squamous cell carcinoma of the pharynx have been observed in the retropharyngeal lymph nodes, the lungs and the pleura (Tokarnia and others, 1969).

In animals affected by upper alimentary squamous cell carcinoma, numerous papillomas have frequently been observed, scattered throughout the upper alimentary tract and occupying the sites in which carcinoma can be found, i.e. the posterior palate, base of the tongue, pharynx, oesophagus and rumen (Plowright, 1955; Curial, 1964; Dobereiner and others, 1967; Tokarnia and others, 1969; Plowright and others, 1971; Pirie, 1973; Campos Neto and others, 1975). In most of these studies, papillomas were found in over 90 per cent of the animals with carcinoma upon which detailed post-mortem examinations were performed. The single exception was the study of Plowright and others (1971) in which papillomas were present in only 50 per cent of cases with carcinoma. Several authors also identified various urinary bladder neoplasms, including haemangiomas and transitional cell carcinomas, in a small proportion of the cattle affected by upper alimentary squamous cell carcinoma, but these animals did not necessarily exhibit clinical signs of enzootic bovine haematuria (Curial, 1964; Dobereiner and others, 1967; Tokarnia and others, 1969; Plowright and others, 1971; Pirie, 1973; Campos Neto and others, 1975).

(2) Urinary Bladder Neoplasia

The syndrome associated with urinary bladder neoplasia is generally known as enzootic bovine haematuria

and is recognised in adult cattle in which it has an insidious onset and, usually, a slow progressive course (Pamukcu, 1955; Beran, 1966). Haematuria is the presenting clinical sign in most animals although initially it is often only observed at the end of micturition (Kalkus, 1913; Hadwen, 1917; Roberts, 1923; Craig, 1930; Pamukcu, 1955). However, the frequency of micturition may be increased and, even at this early stage, straining may occasionally be evident (Kalkus, 1913; Craig, 1930). Subsequently, haematuria becomes apparent throughout micturition but the amounts of blood present can be extremely variable, ranging from small quantities,which tinge the urine pale pink,to larger quantities which result in bright red discolouration of the urine (Roberts, 1923; Bull, Dickinson and Dann, 1932).

Haematuria is frequently intermittent in occurrence during the early stages and may be observed for several days or weeks before remission lasting weeks, months or, in exceptional cases, for as long as a year (Hadwen, 1917; Burnett, 1937; Datta, 1953; Beran, 1966). However, during periods of apparent clinical remission, haematuria can, on occasion, be detected microscopically (Bankier, 1943). Progressively the duration of any remissions shorten and eventually haematuria becomes persistent (Roberts, 1923), as is the

case in some animals from the outset (Hadwen, 1917; Bankier, 1943). With the development of persistent haematuria, the quantity of blood tends to increase and often blood clots are found in the urine (Kalkus, 1913; Bankier, 1943). The severity of haematuria is thought to be exacerbated by several factors including straining,exercise and parturition (Pamukcu, 1955; Beran, 1966) but abatement of haematuria has also been ascribed to the last event (Datta, 1953).

Once haematuria becomes severe and prolonged, there is depression of milk yield (Kalkus, 1931; Bankier, 1943) and loss of weight occurs (Hadwen, 1917; Burnett, 1937; Datta, 1953; Beran, 1966). Pallor of the mucosae develops and,in animals which have become markedly anaemic, oedema of the submandibular space and presternal area may be evident (Hadwen, 1917; Craig, 1930; Beran, 1966; Tokarnia and others, 1969; Smith and Beatson, 1970). Terminally diarrhoea may occur (Hadwen, 1917; Craig, 1930). Occasionally, under management systems in which animals are infrequently handled or closely observed, haematuria passes unnoticed and one of the clinical signs which develops later in the disease process is the initial presenting sign (Smith and Beatson, 1970).

Few authors have described their findings on examination of the urinary bladder by palpation per rectum or per vaginum. Craig (1930) reported that he could detect thickening of the bladder wall by both routes and Butozan and Mihajlovic (1959) state that, in occasional animals, palpation reveals the presence of nodular masses

in the urinary bladder. In addition Gotze (1942) and Rosenberger (1971) have described the visualisation of lesions in the urinary bladder by means of cystoscopy.

The course of the disease tends to be extremely variable depending on the length of remissions and the severity of the haematuria, but most authors agree that it is usually between six months and three years. However, the course can be much shorter, severe anaemia and death occurring within two to three months of the first signs of haematuria (Craig, 1930; Beran, 1966) or, occasionally,much longer with animals surviving for five or six years after haematuria is first observed (Kalkus, 1913; Datta, 1953). Craig (1930) and Pamukcu (1955) record that many owners of affected animals believe that the course of the disease is accelerated by exercise, excitement, pregnancy and poor feeding, and Hadwen (1917) states that in males the course tends to be shortened due to the greater likelihood of urethral obstruction.

The death of affected animals has been ascribed to a variety of causes including,massive haemorrhage from the urinary bladder (Hadwen, 1917; Datta, 1953; Pamukcu, 1955), secondary cystitis and pyelonephritis (Hadwen, 1917; Craig, 1930; Bull and others, 1932; Datta, 1953; Martincic, 1955),and urinary tract obstruction by blood clots resulting in massive uraemia (Hadwen, 1917; Burnett, 1937; Bankier, 1943) hydronephrosis (Pamukcu, 1955) or bladder rupture (Bull and others, 1932). In cases in which the course of the disease has been prolonged, cachexia has

also been cited as a cause of death (Hadwen, 1917; Dickson, 1940; Forero, 1960), but in areas where enzootic bovine haematuria is well recognised, affected animals are frequently culled prior to the terminal stages of the disease (Burnett, 1937; Bankier, 1943).

It is generally agreed that haematological changes only occur in animals which are markedly haematuric and that the changes are typical of a haemorrhagic anaemia (Hadwen, 1917; Craig, 1930; Datta, 1953; Pamukcu, 1955; Forero, 1960; Beran, 1966; Mugera and Nderito, 1968). In these animals, there is a progressive depression of haemoglobin concentration and erythrocyte count which terminally may fall as low as 3-4 g/100ml and 1-2.5 x 10^6/mm^3 respectively (Craig, 1930; Kalkus, 1931; Hess, 1955; Forero, 1960; Beran, 1966; Mugera and Nderito, 1968). The data presented by Forero (1960) indicates that the anaemia is hypochromic and macrocytic. Georgiev (1957) and Singh, Joshi and Prasad (1974) also consider that the anaemia is hypochromic whereas Rosenburger (1971) states that it is usually normochromic. Anisocytosis and poikilocytosis of the erythrocytes have been observed by Kalkus (1931), Datta (1953) and Singh and others (1974) but Georgiev (1957) states that these features do not occur, even in severely haematuric animals. The total and differential leukocyte count is usually within the normal range although leukopaenia may be observed in markedly anaemic animals (Rosenberger, 1971) and,occasionally,there is leukocytosis associated with the neutrophilia of secondary pyogenic infections (Pamukcu, 1955). Affected

animals show no evidence of disturbance of the blood coagulation mechanism (Rosenberger, 1971; Singh and others, 1974).

Changes in the blood biochemistry of animals with enzootic bovine haematuria have rarely been recorded. Singh, Joshi and Ray (1973) and Singh and others (1974) observed that the serum iron, calcium, phosphorus, chloride and albumin were depressed in affected animals and that serum globulin was raised. In contrast, normal values of blood calcium and phosphorus have been recorded by Georgiev (1957) and Forero (1960), and Bankier (1943) did not observe any depression of serum iron in the haematuric animals which he sampled.

Examination of the urine of affected animals can reveal macroscopic, microscopic or no evidence of haematuria, depending on the stage and severity of the disease at the time of sampling. Obvious macroscopic discolouration of the urine by blood may be apparent, and in the latter stages of the disease blood clots are frequently present, in some cases to such an extent that the whole urine sample coagulates on standing (Craig, 1930). With the exception of cases in which the haematuria is very severe, centrifugation produces a clear supernatant urine in which evidence of haemolysis is rarely a feature (Craig, 1930; Rosenberger, 1971). Microscopic examination of the urine sediment reveals the presence of erythrocytes, not only in animals which have clinical haematuria but frequently also in animals in which haematuria is apparently in remission (Beran, 1966). In addition to erythrocytes, various

tissue cells, leukocytes, casts, urinary salts and bacteria may occasionally also be found in the urine sediment (Hadwen, 1917; Craig, 1930; Pamukcu, 1955). Other changes which have been recorded in the urine of affected animals include markedly increased urine protein and elevated levels of urine sugar, calcium and chloride (Singh and others, 1974).

Since the early twentieth century, there has been general agreement that the pathological changes responsible for enzootic bovine haematuria are confined to the urinary bladder and, despite initial confusion as to whether the changes were inflammatory or neoplastic, the overwhelming evidence indicates that the urinary bladder lesions are primarily neoplastic.

The main pathological changes which were described by early workers were the presence of areas of mucosal congestion composed of networks of dilated, thin-walled capillaries and tumour-like lesions including sessile or pedunculated nodules and cauliflower-like growths, many of which also contained highly vascular tissue (Moussu, 1904; Hadwen, 1917; Roberts, 1923; Craig, 1930). Thereafter, on microscopic examination of the urinary bladders of affected animals, Bull and others (1932) confirmed the presence of haemangiomas, papillomas and, in one case, carcinoma. Similar changes were also found by Plummer (1944) and Goto, Kato and Hoshikawa (1954) who described various urinary bladder neoplasms including haemangiomas, papillomas, transitional cell carcinomas and adenocarcinomas.

The first detailed investigations of the pathology and histopathology of urinary bladder neoplasms,which demonstrated the extremely broad spectrum of neoplastic change which can occur in cattle and buffalo affected by enzootic haematuria,were performed by Pamukcu (1955, 1957) in Turkey.

This author described a variety of macroscopic lesions including small congested patches of bladder mucosa, punctate haemorrhagic foci, haemangiomas and papillomas of varying sizes and number, and papillary and non-papillary infiltrating growths, all of which could occur alone or in combination. On microscopical examination tumours of both mesenchymal and epithelial origin were identified. Haemangiomas were most commonly present and constituted approximately 40 per cent of all the urinary bladder tumours found. Papillomas and transitional cell carcinomas accounted for approximately 20 per cent each, and the remaining 20 per cent was composed of a vast range of tumours including adenocarcinomas, adenomas, squamous cell carcinomas, cystadenocarcinoma, and fibrosarcomas. Metastasis was rare and usually extended only to the local lymph nodes, although in one case, a squamous cell carcinoma, secondary deposits were found in the lungs.

Subsequently these findings have been consistently confirmed by workers in numerous countries including Brazil (Dobereiner and others, 1967; Tokarnia and others, 1969), Bulgaria (Sofrenovic, Bratanovic and Stamatovic, 1962), India (Nandi, 1969), Indonesia (Ressang and Sikar, 1960), Japan (Suzuki, 1964; Ito, Miura, Ohshima and Numakunai,

1971), Kenya (Mugera and Nderito, 1968, 1969), New Zealand (Smith and Beatson, 1970) and the Philippines (Beran, 1966). In general, random distribution of neoplastic lesions within the urinary bladder has been reported, but some authors including Craig (1930), Pamukcu (1957) and Mugera and Nderito (1968) state that the lateral and ventral walls are the sites most commonly affected. Occasionally small tumours, usually papillomas, have also been found in the ureters and kidney pelvises (Bull and others, 1932; Pamukcu, 1955).

Inflammatory changes in the urinary tract, which are considered to be secondary to the neoplastic lesions, have frequently been reported in animals affected by enzootic bovine haematuria. These changes include cystitis with hyperplastic and fibrous thickening of the wall of the urinary bladder and pyelonephritis (Craig, 1930; Kalkus, 1931; Bankier, 1943; Beran, 1966; Nandi, 1969).

Lymphosarcoma

Despite the considerable volume of literature devoted to the epidemiology, aetiology and pathology of bovine lymphosarcoma there are very few detailed

descriptions of the clinical aspects of this disease. However it has been established that on the basis of age of the affected animal and anatomical distribution of lesions four clinical syndromes can occur.

Immature animals aged less than two years usually exhibit either the multicentric or thymic forms of the disease (Bendixen, 1961b; Theilen and Madewell, 1979) but on rare occasions a skin form has also been reported (Hugoson, 1966).

Multicentric lymphosarcoma in immature animals occurs most frequently in calves aged less than six months, has occasionally been observed in the foetus, and is considered to be more common in dairy than in beef breeds (Theilen and Madewell, 1979). Generalised superficial lymphadenopathy is the outstanding clinical feature of this form of the disease (Theilen and Dungworth, 1964; Theilen and Madewell, 1979). Most animals also exhibit dullness, weight loss and weakness and less frequently other clinical signs including tachycardia, hyperpnoea, pyrexia, diarrhoea, ruminal tympany and posterior paresis are observed. Of the nine calves examined clinically by Theilen and Dungworth (1964), seven were examined haematologically. Five calves had macrocytic anaemia and three,which had leukocyte counts ranging between 17.5 and 44.3 x 10^3 leukocytes per mm^3,were

considered to have lymphocytic leukaemia. In addition, three calves, one of which was not leukaemic, were found to have atypical lymphocytes in their peripheral blood. Both Theilen and Dungworth (1965) and Theilen and Madewell (1979) reported that serum globulin levels were low in many of the calves affected by multicentric lymphosarcoma which they examined.

Post mortem examinations by these authors revealed generalised lymph node and bone marrow infiltration in every animal. A high proportion of cases (>60%) also had macroscopic involvement of the liver, spleen and kidneys. Other organs which were less frequently affected included the heart, uterus, abomasum and intestine,and in these sites the neoplastic changes were often only recognised microscopically.

Thymic lymphosarcoma is recorded as occurring primarily in animals of beef breeds aged between six and 30 months (Dungworth, Theilen and Lengyel, 1964). These authors described 14 cases in which the major clinical signs were the result of the presence of a large thymic neoplasm in the lower neck or anterior thorax with consequent pressure effects on the respiratory, cardiovascular and alimentary systems. The most common clinical signs observed were distention of the jugular veins, oedema of the presternal area, hyperpnoea or dyspnoea, tachycardia, dullness, loss of weight, fever, anorexia and ruminal tympany. Similar clinical signs are recorded by Theilen and Madewell (1979). Haematological examinations were performed in eight of the animals examined by Dungworth

and others (1964) of which four were anaemic and one had lymphatic leukaemia.

Detailed post mortem examinations performed by Dungworth and others (1964) and Theilen and Madewell (1979) revealed total or partial replacement of the thymus by tumour tissue in every animal. In the majority of cases (>90%) there was involvement of lymph nodes, particularly the broncho-mediastinal and superficial cervical nodes, but generalised lymph node infiltration was never observed. Extensive involvement of other organs was seldom present and infiltration of the liver, spleen kidneys or heart was confined to less than 30 per cent of cases.

The skin form of lymphosarcoma appears to be uncommon compared with the other clinical forms of the disease and, although it has been reported in all ages of cattle, most cases have been described in adults particularly those aged around three years rather than immature animals (Bendixen, 1961b; Theilen and Madewell, 1979). Affected animals present with rounded nodules or plaques in the skin which usually have a widespread distribution over the body although they tend to be particularly numerous on the neck, back, flanks and thighs (Bendixen, 1961b). The lesions may ulcerate and become necrotic. In addition to the skin lesions there is frequently enlargement of all the superficial lymph nodes and there may be clinical evidence of involvement of other organs, although this has never been described in detail (Bendixen, 1961b; Theilen and Madewell, 1979).

Several authors including Bendixen (1961b) and Clegg and Moss (1965) have described regression of the skin plaques within a period of weeks or months of their initial appearance. Complete healing of the skin and regrowth of hair occurs with regression of the lesions and, in some cases in which the superficial lymph nodes are enlarged, these also regress to normal size. However clinical evidence of lymphosarcoma recurs with the development of lesions in the skin and, or other organs over a period ranging between several weeks and three years.

The distribution of lesions other than those in the skin and lymph nodes are poorly documented, although involvement of the heart, liver, spleen, kidneys and spinal canal has been reported (Bendixen, 1961b; Clegg and Moss, 1965). Haematological and biochemical changes have not been described in animals affected by the skin form of lymphosarcoma.

In adult cattle, lymphosarcoma usually presents as the multicentric form of the disease and the highest prevalence is seen amongst dairy cattle aged between four and eight years (Marshak, Coriell, Lawrence, Croshaw, Schryver, Altera and Nichols, 1962; Bendixen, 1961a). The main clinical feature is enlargement of the superficial lymph nodes particularly the mandibular, superficial cervical and sub-iliac nodes. In addition, the lymph nodes of the abdominal cavity, including the medial iliac nodes, are frequently found to be palpably enlarged on rectal examination. However, in contrast with calves affected by

multicentric lymphosarcoma, generalised, bilaterally symmetrical lymphadenopathy is only seen in approximately 50-60 per cent of cases and the remainder have enlargement of individual or localised groups of lymph nodes (Bendixen, 1961a; Marshak and others, 1962).

The majority of animals show loss of condition, partial or total anorexia, decreased milk production and many exhibit clinical signs associated with enlargement of lymph nodes and infiltration of organs within the thoracic or abdominal cavities. Cardiovascular abnormalities are commonly identified. Tachycardia, pallor of the mucous membranes and distention of the jugular veins are found in a high proportion of cases but cardiac murmurs and arrhythmias are less frequently encountered. Mild respiratory signs including hyperpnoea and tachypnoea are often present and in a few cases percussion of the thorax reveals an area of decreased resonance. Diarrhoea is occasionally evident but other clinical signs referable to the alimentary and urinary tracts are seldom apparent.

Posterior paresis and exophthalmus due to the presence of a retrobulbar tumour mass are two prominent clinical signs which are recognised in approximately 30 per cent of adults with multicentric lymphosarcoma although they are rarely found in other forms of the disease. Pyrexia is also a relatively common feature and may be present in up to 40 per cent of affected animals.

It has been reported that, on rare occasions, sudden deaths can occur in adult animals with multicentric lymphosarcoma prior to the development of any other clinical signs. These deaths have been attributed to rupture of the spleen, acute heart failure and perforation of tumorous ulcers of the abomasum with resultant peritonitis (Bendixen, 1961a; Theilen and Madewell, 1979).

Haematological examination of affected animals commonly reveals the presence of a normocytic, normochromic anaemia (Gotze, Rosenberger and Ziegenhagen, 1954; Marshak and others, 1962) which tends to be most severe in animals which are also leukaemic (Marshak and others, 1962). Many adult cattle with lymphosarcoma are found to have high leukocyte counts as a result of either frank leukaemia or lymphocytosis due to a marked increase in the numbers of immature and abnormal lymphocytes (Theilen and Madewell, 1979). However, Marshak and others (1961) stress that in many cases leukocyte counts lie within the normal range, and the occasional detection of abnormal lymphocytes in these animals is of little significance as they can be found in healthy cattle.

On post mortem examination, the sites most frequently involved are the lymph nodes, heart, abomasum, kidneys and uterus (Bendixen, 1961a; Marshak and others, 1962; Theilen and Madewell, 1979). Involvement of the lymph nodes is recognised in over 90 per cent of animals and the distribution of lymphadenopathy may be generalised and bilaterally symmetrical or confined to

localised groups of nodes. Heart lesions which are thought to arise in the right atrium (Jarplid, 1964; Dungworth, Thielen and Ward, 1968) are particularly common and may be found in up to 90 per cent of cases. Abomasal, renal and uterine involvement occurs in over 50 per cent of animals whereas hepatic and splenic infiltration is less frequently recognised, particularly in comparison with the multicentric form of calves, and is variously reported as being present in between 16 and 58 per cent and 27 and 40 per cent of cases respectively (Bendixen, 1961a; Marshak and others, 1962; Theilen and Madewell, 1979). Other major sites of tumour infiltration are the epidural space in which lymphosarcomatous tissue can be found in approximately 50 per cent of animals and the intestines, omasum, reticulum and rumen, one or more of which can be involved in excess of 45 per cent of cases. In addition, the retrobulbar space and the urinary bladder are occasionally affected but infiltration of the lungs or udder is rare.

Although adult cattle with lymphosarcoma usually present with the multicentric form of the disease, Bendixen (1961b) has recorded that the skin form is not uncommon amongst this age group. The clinical signs and pathological features are similar to those found in immature animals.

A CLINICO-PATHOLOGICAL STUDY OF UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA, URINARY BLADDER NEOPLASIA AND LYMPHOSARCOMA IN CATTLE

INTRODUCTION

The results of the survey of bovine neoplasia described in Chapter 1 indicate that, within the geographical confines of the survey, upper alimentary squamous cell carcinoma, malignant and benign urinary bladder neoplasia and lymphosarcoma are the most frequently encountered neoplasms which are likely to be of economic significance, in that, they will cause loss of production and, eventually, a fatal outcome in affected animals.

Despite the considerable volume of literature related to both urinary bladder neoplasia and lymphosarcoma, there are few detailed clinico-patholigical descriptions of either condition. This is particularly evident with urinary bladder neoplasia as authors have tended to concentrate on either the clinical or pathological aspects in isolation, but a similar situation exists as regards lymphosarcoma with the notable exceptions of the studies performed by Theilen and Marshak and their respective co-workers in the United States and Bendixen in Denmark. In respect of upper alimentary squamous cell carcinoma, there is a paucity of either clinical or pathological data, and descriptions, in any detail, are almost entirely confined to the work of Dobereiner, Campos Neto and their respective co-workers in Brazil and Plowright in Kenya.

The following study was performed in order to provide a detailed clinical appraisal of these neoplasms which could be related to the pathological findings and used as a template for accurate diagnosis in the field.

MATERIALS AND METHODS

(1) Animals

The animals were those of the cases in Chapter 1 which had upper alimentary squamous cell carcinoma, urinary bladder neoplasia or lymphosarcoma and, upon which it was possible to perform a detailed clinical examination.

(2) Clinical Examination and Terms

A detailed physical examination was carried out on admission of the animal to the Veterinary Hospital and, unless otherwise stated, all results refer to this initial examination. Samples of blood, faeces and urine, as considered necessary, were also taken on admission.

The following clinical terms have been used which require definition. Tachypnoea was considered to be present when the resting respiratory rate was greater than 30 respirations per minute and tachycardia when the resting heart rate was greater than 90 beats per minute. Hyperpnoea was considered to be present when it was obvious that the abdominal muscles were being used at rest to assist respiration but dyspnoea was reserved for respiratory distress when an animal was seen to be mouth-breathing and heard to grunt on expiration. Pyrexia is used to describe a rectal temperature in excess of 102.5°F. Cud-dropping is the term applied to the phenomenon in which an animal, in the normal process of rumenation, regurgitates a bolus of ingesta in order to cud but cannot retain the bolus within its mouth and allows it to drop to the ground. The lymph node nomenclature utilised is that of Nomina Anatomica Veterinaria (1973).

(3) Haematology

After blood had been collected in bottles containing ethylene diamine tetra-acetic acid, the packed cell volume was measured by the microhaematocrit technique (Fisher, 1962), the amount of haemoglobin was estimated using the oxyhaemoglobin method (Dacie and Lewis, 1963) and the total numbers of erythrocytes and leukocytes were counted using a model D Coulter counter (Coulter Electronics Ltd., Dunstable, Beds.). The differential leukocyte counts were made on Leishman-stained blood films and 200 cells were counted.

The mean cell volume (MCV) expressed as cubic microns was calculated from the formula

$$MCV = \frac{\text{Packed cell volume (\%) x 10}}{\text{Erythrocyte Count } (x10^6/mm^3)}$$

and the mean cell haemoglobin concentration (MCHC) expressed as a percentage from the formula

$$MCHC = \frac{\text{Haemoglobin (g/100ml) x 100}}{\text{Packed cell volume (\%)}}$$

The normal haematological values recorded are those quoted by the Department of Veterinary Pathology of Glasgow University Veterinar Hospital.

(4) Blood Biochemistry

After blood had been collected in heparinised tubes the urea, bilirubin, inorganic phosphate, alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase and the total protein values were measured using a Technicon auto-analyser (Technicon Instrument Corporation, Tarry Town, New York, U.S.A.). The potassium and sodium values were estimated using an EEL flame photometer, chloride was measured using an EEL chloride meter, the calcium and magnesium values were found using a Unicam S.P.90 (Pye-Unicam,

Cambridge, England). The amounts of albumin and globulin were measured using zone electrophoresis scanned with a Kipp and Zonen microdensitometer. In diarrhoeic animals only, the serum pepsinogen values were estimated by the method of Edwards, Jepson and Wood (1960) and expressed as milli-international units of tyrosine (milli-mols tyrosine per litre plasma per minute x 1,000). The normal biochemical values recorded are those quoted by the Department of Veterinary Biochemistry of Glasgow University Veterinary Hospital.

(5) Examination of Faecal Samples

Fresh faeces from diarrhoeic animals were examined for the presence of trichostrongyle eggs by a modification of the McMaster technique of Gordon and Whitlock (1939) and in animals aged over two years for the presence of clumps of acid-fast bacteria which morphologically resembled Mycobacterium paratuberculosis by the method of Cunningham and Gilmour (1959). A positive result was recorded if clumps of acid fast bacilli were found. When only single organisms were identified, this was considered an inconclusive result and at least one repeat sample of faeces was examined.

(6) Examination of Urine Samples

The amount of urine protein was estimated by the sulphosalicylic acid precipitation test as described by Kingsbury, Clarke, Williams and Post (1926).

(7) Statistical Methods

The statistical methods used were the "Student's" t test and the coefficient of correlation (Bishop, 1971). Unless otherwise stated, when a difference is described as

significant, this implies that the probability of its resulting from chance is less than two per cent ($p = < 0.02$) and when a difference is described as highly significant this implies that the probability of its resulting from chance is less than 0.1 per cent ($p = < 0.001$).

SECTION I

CLINICAL ASPECTS OF UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

RESULTS

Clinical examination of animals which were subsequently pathologically confirmed to be affected by squamous cell carcinoma of the upper alimentary tract revealed that the majority of cases could be attributed to one of four clinical syndromes;

(i) an oropharyngeal syndrome which is characterised clinically by poor body condition, halitosis, dribbling of saliva, coughing, snoring and the presence of an oropharyngeal mass,

(ii) an oesophageal syndrome which is characterised clinically by poor body condition, cud-dropping, the presence of a mass in the cervical oesophagus, halitosis and diarrhoea,

(iii) a ruminal tympany syndrome characterised by poor body condition, rumenal tympany and profuse diarrhoea, and,

(iv) a wasting and diarrhoea syndrome characterised clinically by poor body condition and profuse diarrhoea.

In addition a small number of animals exhibited clinical signs which were sufficiently different from the majority of the cases that they could not be attributed to any one of the four syndromes and thus had to be considered as individual entities.

(i) Oropharyngeal Syndrome

Case Histories

A history was available for eight of the 16 animals. Progressive loss of condition over a period of three weeks to six months had been observed in all the animals and subsequently coughing and, or drooling of saliva had developed in six cases. The loss of condition was associated with chronic intermittent diarrhoea in one of the two remaining animals but in the other the owner had not observed any additional features. All 16 animals were female and were aged between eight and 14 years. The individual case histories are summarised in Table 6.

Presenting Signs

Dribbling of saliva (Figure 15) and, or snoring and, or coughing were the main presenting signs in all but one case (A3), in which the major feature was a large mandibular swelling. Poor bodily condition was ubiquitous. Occasionally profuse diarrhoea (A4, A12, A13) or dyspnoea (A8) were additional presenting signs.

Clinical Signs

The major clinical findings for each individual animal are summarised in Table 7.

All the animals were in poor bodily condition and afebrile. The majority (63%) were dull.

Significant clinical signs were mainly confined to the alimentary and upper respiratory tracts. Halitosis and drooling or dribbling of saliva were virtually ubiquitous, being present in 100 per cent and 94 per cent of the animals respectively. An oropharyngeal mass was detected visually

TABLE 6

Oropharyngeal Syndrome

Summary of Case Histories

Case Number	Breed	Age	Loss of Condition	Coughing	Drooling Saliva	Other
A1	Sh x	9y		No history available		
A2	Sh x	>10y		No history available		
A3	High	>10y		No history available		
A4	Gall	8y		No history available		
A5	AA x	>10y		No history available		
A6	AA x	>10y		No history available		
A7	Gall x	>10y		No history available		
A8	High x	>10y	Two months	Six days	Two days	
A9	High	14y	Two months		One week	
A10	Sh	8y	One month	One week	One week	
A11	Sh x	>10y		No history available		
A12	High	12y	One month	One month		
A13	AA	>8y	Six months			Intermittent diarrhoea for six months
A14	AA	12y	Two months	Two weeks		
A15	AA	>10y	Three weeks	Three weeks	Three weeks	
A16	AA	10y	Three months			

TABLE 7

Oropharyngeal Syndrome

Summary of Major Clinical Findings

Case Number	Body Condition	Demeanour	Dribbling Saliva	Halitosis	Oropharyngeal Mass	Oropharyngeal Papillomas	Coughing	Snoring	Nasal Discharge Containing Ingesta	Enlargement Submandibular Lymph Nodes	Diarrhoea
A1	Poor	Dull	Present	Present	Small	Few	Frequent	Present	-	-	Present
A2	Poor	Bright	Marked	Present	Small	Numerous	-	-	-	-	Present
A3	Very poor	Dull	Present	Present	Large	-	-	-	-	Slight	-
A4	Poor	Bright	Marked	Present	Large	Numerous	Occasional	-	-	Slight	Profuse
A5	Poor	Dull	Marked	Marked	Large	Few	Occasional	-	-	Marked	-
A6	Poor	Dull	-	Marked	-	Few	Frequent	Present	-	-	-
A7	Poor	Dull	Present	Marked	Large	Few	Frequent	Present	Present	-	Profuse
A8	Very poor	Dull	Marked	Marked	-	-	Occasional	Present	-	-	-
A9	Poor	Dull	Present	Marked	Small	-	-	Present	Present	-	-
A10	Poor	Dull	Present	Marked	Large	-	Occasional	Present	Present	-	-
A11	Poor	Dull	Marked	Present	Small	Solitary	-	-	-	Marked	-
A12	Poor	Dull	Present	Marked	Small	Numerous	Frequent	-	Present	-	Profuse
A13	Poor	Bright	Present	Marked	Small	Numerous	-	Present	-	Slight	Profuse
A14	Poor	Bright	Marked	Marked	Large	Numerous	Occasional	Present	Present	Slight	-
A15	Very poor	Bright	Present	Present	Large	Numerous	Frequent	-	Present	Slight	-
A16	Poor	Bright	Marked	Marked	Large	Few	Frequent	-	Present	Slight	Profuse

and,or by palpation in 14 animals (87%) affecting the pharynx in 50 per cent, the posterior body and, or base of the tongue in 50 per cent and the smooth part of the hard palate and, or soft palate in 25 per cent. In general the lesion was a raised, irregular, fungating mass, the surface of which was ulcerated and necrotic, but in two cases (A4, A5) the lesion took the form of a deep, eroding, necrotic ulcer involving the posterior body of the tongue. No major oropharyngeal lesion could be found in two animals (A6, A8) but in the latter a mass was detected in the oesophagus extending distally 12 cms from the level of the larynx, and which completely obstructed the passage of a stomach tube.

Extension of the oropharyngeal lesion to involve surrounding structures was evident in two animals (A3, A5). In the former, a mass 8 cms in diameter involving the masseter muscle was present at the angle of the ramus of the right mandible, and in the latter a firm swelling involving the mylohyoid muscle was present in the intermandibular space. In addition submandibular lymph node enlargement, suggestive of metastasis was detected in 50 per cent of the animals.

Examination of the oropharynx also revealed the presence of papillomas, as distinct from the lesions already described, in 75 per cent of the animals, most frequently situated on the smooth part of the hard palate and, or soft palate and less commonly on the tongue or pharyngeal walls.

Although only six animals (38%) had obvious difficulties in mastication and deglutition, the majority (69%) had a reduced appetite. Other abnormalities referrable to the alimentary tract were diarrhoea, present in seven animals (44%) and developing post admission in a further four (A10, A11, A14, A15), and reduced intensity of ruminal contraction, evident in seven (44%). Reduced abdominal volume was a feature in five animals (31%).

Respiratory signs were a feature in 14 animals (88%) but in most were confined to the upper respiratory tract. Coughing and snoring were the most prominent signs occurring in 11 animals (69%) and eight animals (50%) respectively, with each developing in a further one animal (A13, and A12) post admission. Ten animals (63%) had a mucopurulent nasal discharge (Figure 15), which in seven (44%) contained ingesta, a feature most marked when coughing was also present. In addition, fresh blood was observed in the nasal discharge of two animals (A8, A16). Dyspnoea, characterised by mouth breathing and the adoption of an "air hunger" position was an occasional clinical sign being observed in three animals (A1, A6, A8), although in the first two it developed only after mild exercise.

Signs referable to the lower respiratory tract were uncommon. Seven animals (44%) were hyperpnoeic, three of which had harsh respiratory sounds, and two rhonchi on auscultation, but only one animal was tachypnoeic.

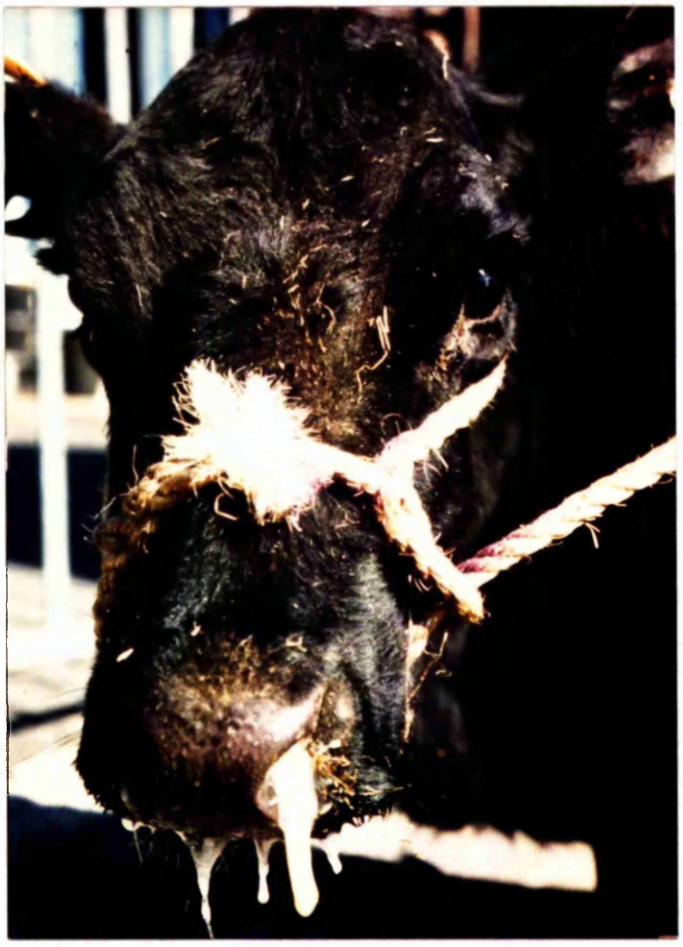


FIGURE 15 Oropharyngeal syndrome : dribbling of saliva and mucropurulent nasal discharge

Other significant clinical signs were confined to the cardiovascular and urinary systems. Pallor of the mucosae was evident in three animals (A4, A9, A11) and developed in a further three (A12, A13, A14) post admission. Haematuria was observed in two animals (A2, A12).

Haematology

The results of haematological examinations performed on individual animals on admission are recorded in Appendix 3. The mean values of packed cell volume, haemoglobin concentration and erythrocyte count are at the lower extreme of the normal range (Table 8) but only two animals (A3, A4) were anaemic (packed cell volume <25% and haemoglobin concentration <8g/100mls). The mean values of mean cell haemoglobin concentration and mean cell volume are also within the normal range.

The mean total leukocyte count was within the normal range (Table 8). Three animals (A1, A6, A12) had marked leukocytosis (>13.0 x 10^3 leukocytes/mm^3),which was due to neutrophilia in each case,and one animal (A16) had marked leukopaenia (<3.5 x 10^3 leukocytes/mm^3) due to both neutropaenia and lymphopaenia.

Biochemistry

The results of blood biochemical analysis performed on each animal on admission are recorded in

TABLE 8

Oropharyngeal Syndrome

Summary of Haematological Parameters

on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	16	28.5 ± 4.7	27 - 35
Haemoglobin (g/100mls)	14	9.1 ± 1.1	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	14	5.19 ± 1.03	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	14	32.8 ± 2.0	30 - 36
Mean Cell Volume (μ^3)	14	55.2 ± 8.1	40 - 60
Leukocyte Count ($x10^3/mm^3$)	16	9.0 ± 3.8	7 - 10

Appendix 3. Abnormal values of plasma albumin and globulin were found in a high proportion of the animals with the result that the mean value for plasma albumin was markedly depressed and the mean value for plasma globulin was raised (Table 9). Plasma albumin was low (<25g/l) in all but one of the animals (A15) and was markedly depressed (<20g/l) in seven cases (44%). Plasma globulin was raised (>55g/l) in all but one of the animals (A4) and was markedly elevated (>65g/l) in nine cases (56%).

In addition, the mean value of plasma magnesium was outwith the lower limits of the normal range (Table 9) and eight of the 12 animals in which this parameter was measured had low values. The mean values of the other plasma constituents which were measured were within their normal ranges (Table 9) and in individual animals markedly abnormal values were infrequently encountered.

Faecal Examination

On admission, the faeces of all seven diarrhoeic animals were examined for the presence of nematode eggs and acid fast bacilli resembling Mycobacterium paratuberculosis. The results for each individual animal are recorded in Appendix 3.

Trichostrongyle eggs were identified in four animals (A2, A7, A12, A16) which had counts of 50, 200, 5800 and 200 eggs per gram (e.p.g.) of faeces respectively. The animal with 5800 epg was treated with thiabendazole (Thibenzole suspension^{R} - Merck, Sharpe and Dohme Limited)

TABLE 9

Oropharyngeal Syndrome

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	16	6.2 ± 8.1	0 - 8.3
SODIUM mmol/l	15	137 ± 6	136 - 151
POTASSIUM mmol/l	15	4.4 ± 1.2	3.2 - 5.8
CHLORIDE mmol/l	14	96 ± 7	96 - 111
CALCIUM mmol/l	12	2.43 ± 0.33	2.29 - 3.08
MAGNESIUM mmol/l	12	0.59 ± 0.29	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	16	1.69 ± 0.44	1.13 - 2.84
BILIRUBIN µmol/l	15	5 ± 3	0 - 8
ALKALINE PHOSPHATASE IU/l	15	41 ± 28	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	16	129 ± 77	0 - 200
ALANINE AMINOTRANSFERASE IU/l	16	25 ± 9	0 - 40
TOTAL PROTEIN g/l	16	87 ± 12	50 - 90
ALBUMIN g/l	16	19 ± 5	25 - 40
GLOBULIN g/l	16	68 ± 9	25 - 55

at the recommended dosage rate and within two days the nematode egg count had fallen to zero but the diarrhoea persisted. The results of faecal examination for the presence of acid fast bacilli resembling M. paratuberculosis were negative in four animals and inconclusive in three animals (A7, A12, A13). However repeat examinations in these latter cases all proved negative.

Serum Pepsinogen Estimation

Serum pepsinogen estimations were performed on admission in five of the seven diarrhoeic animals (Appendix 3). In three animals the serum pepsinogen was less than 2000 mU tyrosine and in the remaining two (A2, A12) the values were 2560 and 2072 mU tyrosine respectively. In these two latter animals the serum pepsinogen levels had fallen to 1800 and 1563 mU tyrosine respectively two weeks post admission, but diarrhoea persisted.

Urine Examination

With one exception (A5) urine from each animal was examined on admission for the presence of protein and erythrocytes (Appendix 3). The urine protein was raised (>50mg/100ml) in five animals of which two (A8, A16) had marked proteinuria. Microscopic examination revealed the presence of erythrocytes in three animals (A2, A9, A12) two of which (A2, A12) were clinically haematuric and all three of which had raised urine protein levels.

Pathological Findings

The oropharynx was the site of the major focus of carcinoma in all but two cases (A6, A8) in which the proximal extremity of the oesophagus was involved. Within the oropharynx, the sites affected were the walls of the pharynx, the posterior body and base of the tongue, the soft palate and the smooth part of the hard palate (Figure 16). The lesion extended to involve two or more of these sites in most of the animals, and in two cases there was massive infiltration of adjacent musculature, viz. the right masseter muscle in case A3 and the mylohyoid muscles in case A5. In cases A6 and A8, in which the major focus of carcinoma involved the proximal extremity of the oesophagus, the neoplasm was locally invasive and had, by direct extension, penetrated the walls of the larynx and trachea respectively.

Metastasis was evident in eight animals (50%) but spread was only local, with metastatic carcinoma confined to the retropharyngeal (A3, A4, A5, A10, A12, A13, A16), mandibular (A3, A4, A5, A16) and parotid lymph nodes (A9, A10).

Other discrete primary foci of upper alimentary squamous cell carcinoma were evident in 11 animals (69%), most commonly situated in the oesophagus. The majority of these foci comprised small (<3cm diameter) fungating or ulcerative lesions, but in three animals (A4, A11, A14) large carcinomas were present on the dorsal non-pigmented area of the rumen.

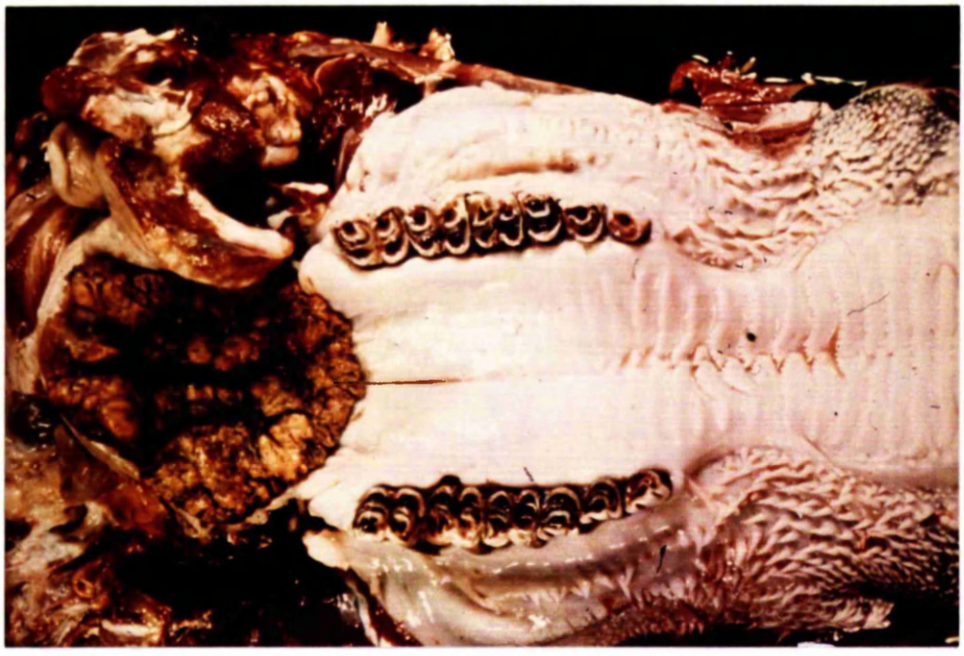


FIGURE 16 Squamous cell carcinoma of the palate and pharynx

Upper alimentary papillomas were found in every animal except case A8. Twelve animals had oropharyngeal papillomas and with the exception of case A8 all had papillomatosis of the oesophagus and/or rumen.

Examination of the lower alimentary tract revealed the presence of adenomas and adenomatous hyperplasia in the small intestines and colons of seven animals (A9, A10, A11, A12, A13, A14, A16) and in case A6, an adenocarcinoma of the distal duodenum which had metastasised to the mesenteric lymph nodes.

Urinary bladder neoplasia was identified in two animals, a solitary haemangioma being found in case A2 and several small transitional cell carcinomas in case A9. This animal (A9) was also found to have an adenoma of the left renal cortex and a melanoma in the pelvic connective tissue.

(ii) Oesophageal Syndrome

Case Histories

A history was available for ten of the 11 animals. Progressive loss of condition over a period of two weeks to several months had been observed in all the animals and subsequently cud dropping had become a feature in seven cases. Two of the remaining cases had developed a swelling in their necks and one had persistent diarrhoea. All 11 animals were female and were aged between eight and 18 years. The individual case histories are summarised in Table 10.

Presenting Signs

Diarrhoea and, or cud dropping and, or the presence of a swelling in the neck were the presenting signs in all the animals except one (B9) which presented with ventral abdominal distention. Poor body condition was a ubiquitous additional presenting sign.

Clinical Signs

The major clinical findings for each individual animal are summarised in Table 11.

All the animals were in poor bodily condition and although all were afebrile, the majority (64%) were of dull demeanour.

Significant clinical findings were mainly referable to the alimentary tract. Cud dropping was a feature in nine animals (82%) and occurred during regurgitation when the animal appeared unable to retain the bolus within the mouth and dropped it to the ground. In addition, two

TABLE 10

Oesophageal Syndrome

Summary of Case Histories

Case Number	Breed	Age	Loss of Condition	Cud Dropping	Other
B1	Gall	12y	Several months	3 weeks	
B2	High	>10y	No history available		
B3	Gall	14y	One month		Swelling in neck observed one week prior to admission
B4	Sh X	13y	One month		Persistent diarrhoea for one month
B5	AA X	8y	Several months	One week	
B6	Sh	15y	Several months	Ten days	
B7	AA	9y	Two weeks	Three days	Rumen tympany observed on one day, three days prior to admission
B8	AA	15y	One month	Ten days	
B9	AA	18y	Two months	One week	
B10	High	16y	Two months		Swelling in neck observed one week prior to admission
B11	AA	8y	Several months	Two days	

TABLE 11

Oesophageal Syndrome

Summary of Major Clinical Findings

Case Number	Body Condition	Demeanour	Cud Dropping	Cervical Mass	Oropharyngeal Papillomata	Diarrhoea	Halitosis
B1	Poor	Bright	Occasional	Palpable	-	Present	-
B2	Poor	Dull	Occasional	Palpable	-	-	-
B3	Poor	Dull	Frequent	Visible	-	Profuse	-
B4	Poor	Bright	-	Palpable	Few	Profuse	Marked
B5	Poor	Bright	-	Palpable	Few	Present	-
B6	Poor	Dull	Occasional	-	Solitary	Present	Present
B7	Poor	Dull	Frequent	Palpable	Numerous	Profuse	Marked
B8	Poor	Dull	Occasional	Palpable	Numerous	-	Present
B9	Poor	Dull	Occasional	-	Numerous	-	Present
B10	Poor	Bright	Occasional	Visible	-	Profuse	Marked
B11	Very poor	Dull	Frequent	-	Numerous	-	Present

TABLE 11 (continued)

Oesophageal Syndrome

Summary of Major Clinical Findings

Case Number	Dribbling of Saliva	Coughing	Abdomen Size	Gurgling from Oesophagus	Obstruction to Passage of Stomach Tube
B1	Present	-	Reduced	-	Marked
B2	-	Occasional	Reduced	Present	Not attempted
B3	Present	Occasional	Reduced	Present	Not attempted
B4	-	-	Reduced	Present	-
B5	-	Occasional	Reduced	Present	Complete
B6	-	-	-	-	-
B7	-	Frequent	Reduced	-	Complete
B8	Present	Frequent	-	-	-
B9	-	-	Distended	Present	Complete
B10	Present	-	Distended	-	-
B11	Present	Frequent	Reduced	-	Complete

animals (B1, B5) were never observed cudding, two (B2, B10) had marked difficulty in swallowing hay, and the latter of these (B10) occasionally "vomited" large quantities of fluid rumen contents. A mass involving the cervical oesophagus was detected by palpation in eight animals (73%), located, with one exception (B10), in the proximity of the left jugular furrow, and situated in the upper third of the neck in cases B4, B7 and B8, the middle third in cases B1, B2 and B3, and in the lower third in case B5. The mass was also situated in the lower third of the neck in case B10 but in this case occupied a ventral midline position. In two animals (B3, B10) the mass was evident on visual inspection but in the third case (B5) in which a swelling in the neck was a presenting sign it was due to fluid distention of the oesophagus proximal to a mass. Peristaltic waves could be observed in the distended oesophagus and these were accompanied by fluid gurgling noises. Similar noises were heard in four other animals (B2, B3, B4, B9) although accumulation of fluid in the oesophagus was not obvious. Passage of a stomach tube was attempted in nine animals and obstruction to passage of the tube was encountered at the level of the palpable mass in three (B1, B5, B7). In a further two (B9, B11) in which no mass had been detected the tube could not be passed beyond obstructions in the mid and upper cervical oesophagus respectively.

As in the oropharyngeal syndrome, halitosis was common, occurring in seven animals (64%), but dribbling of saliva, although present in five animals (45%), was not a prominent feature. Examination of the oropharynx revealed

the presence of papillomas in seven animals (64%). In one of these (B4) the posterior dorsum of the tongue was firm and ulcerated and the submandibular lymph nodes were enlarged. Diarrhoea was also evident in seven animals (64%) and developed post-admission in one further case (B8). An unusual feature of the faeces of four of the diarrhoeic animals (B5, B6, B7, B10) was the presence of long (1-3 cm) fibres of undigested hay. Abdominal volume was reduced in seven animals (64%) but in two (B9, B10) there was bilateral ventral abdominal distention, although no evidence of ruminal tympany. Subsequently the distention disappeared in case B10, whereas in case B11 which initially had a reduced abdominal volume, mild ruminal tympany and abdominal distention developed post-admission. On auscultation, the intensity of ruminal contractions was reduced in seven animals (64%) and in many of these the contractions were irregular or infrequent in occurrence. A variable degree of anorexia was present in a similar number of animals.

In general, upper respiratory signs were less common than in the oropharyngeal syndrome. Frequent coughing was a feature in three animals (B7, B8, B11), in all of which the major lesion was identified as being in the upper cervical oesophagus. During coughing, one (B8) commonly expelled ingesta from its mouth and in another (B7) ingesta could be seen in a nasal discharge after bouts of coughing. Three other animals (B2, B3, B5) coughed occasionally but snoring did not occur in any of the cases. Clinical abnormalities of the lower respiratory tract were

rare. Tachypnoea was recorded in two animals (B1, B6) which had respiratory rates of 50 and 40 per minute respectively, three animals (B1, B2, B6) were slightly hyperpnoeic, and on auscultation, three (B6, B10, B11) had harsh respiratory sounds. Adventitious sounds were not detected in any animal.

Examination of the cardiovascular system revealed pallor of the mucosae in six animals (55%) and one of these (B1) subsequently developed an haemic systolic murmur and subcutaneous oedema.

None of the animals had clinically detectable abnormalities of the urogenital system.

Haematology

The results of haematological examinations performed on individual animals on admission are recorded in Appendix 3 . The mean values of packed cell volume, haemoglobin concentration and erythrocyte count are at the lower extreme of the normal range (Table 12) and four animals (B1, B4, B6, B10) were anaemic. The anaemia was normocytic, and normochromic in three animals and macrocytic and normochromic in the remaining case (B1).

The mean total leukocyte count was within the normal range (Table 12). One animal (B4) had marked leukocytosis (>13.0 x 10^3 leukocytes/mm^3) which was due to

TABLE 12

Oesophageal Syndrome

Summary of Haematological Parameters

on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	11	27.7 ± 7.5	27 - 35
Haemoglobin (g/100mls)	9	8.9 ± 2.6	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	9	4.89 ± 1.50	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	9	32.4 ± 1.7	30 - 36
Mean Cell Volume (μ^3)	9	56.7 ± 5.1	40 - 60
Leukocyte Count ($x10^3/mm^3$)	11	7.2 ± 4.7	7 - 10

a massive neutrophilia and another had marked leukopaenia ($<3.5 \times 10^3$ leukocytes/mm^3) due to both neutropaenia and lymphopaenia.

Biochemistry

The results of blood biochemical analysis performed on each animal on admission are recorded in Appendix 3. Abnormal values of plasma albumin and globulin were found in a high proportion of the animals with the result that the mean value for plasma albumin was markedly depressed in the mean value for plasma globulin was raised (Table 13). Plasma albumin was low (<25g/l) in all but two of the animals (B5, B11) and was markedly depressed in seven cases (64%). Plasma globulin was raised (>55g/l) in all but two of the animals (B2, B5) but was only markedly elevated (>65g/l) in four cases (36%).

In addition, the mean value of plasma calcium was marginally outwith the lower limits of the normal range (Table 13) and low values of this parameter were found in five animals. The mean values of the other plasma constituents which were measured were within their normal ranges (Table 13) and in individual animals markedly abnormal values were infrequently encountered (Appendix 3).

Faecal Examination

On admission the faeces of all seven diarrhoeic animals were examined for the presence of nematode eggs and acid fast bacilli resembling M.paratuberculosis.

TABLE 13

Oesophageal Syndrome

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	11	3.9 ± 1.9	0 - 8.3
SODIUM mmol/l	11	138 ± 5	136 - 151
POTASSIUM mmol/l	11	4.0 ± 0.5	3.2 - 5.8
CHLORIDE mmol/l	11	100 ± 5	96 - 111
CALCIUM mmol/l	11	2.27 ± 0.16	2.29 - 3.08
MAGNESIUM mmol/l	11	1.24 ± 0.62	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	11	1.41 ± 0.49	1.13 - 2.84
BILIRUBIN µmol/l	11	8 ± 6	0 - 8
ALKALINE PHOSPHATASE IU/l	11	61 ± 40	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	11	162 ± 104	0 - 200
ALANINE AMINOTRANSFERASE IU/l	11	24 ± 12	0 - 40
TOTAL PROTEIN g/l	11	82 ± 8	50 - 90
ALBUMIN g/l	11	20 ± 7	25 - 40
GLOBULIN g/l	11	62 ± 9	25 - 55

The results for each individual animal are recorded in Appendix 3.

Trichostrongyle eggs were identified in five animals (B1, B3, B4, B6, B7) which had counts ranging from 50 to 300 epg of faeces. The results of faecal examination for the presence of acid fast bacilli were negative in all seven animals.

Serum Pepsinogin Estimation

Serum pepsinogin estimations were performed on admission in all seven diarrhoeic animals (Appendix 3). None of the animals had a serum pepsinogin in excess of 2000 mU tyrosine.

Urine Examination

Urine from each animal was examined on admission for the presence of protein and erythrocytes (Appendix 3). Urine protein was raised (>50mg/100mls) in one animal (B5) which had a value of 53mg/100mls. Microscopic examination failed to reveal the presence of erythrocytes in the urine of any of the animals.

Pathological Findings

The cervical oesophagus was the site of the major focus of carcinoma in every case, and in the majority (73%) the lesion was situated in the proximal 30 cms. Only two animals had foci of carcinoma in the oropharynx. In case B5 the oesophageal lesion extended to involve the posterior pharynx and there was a discrete focus of

carcinoma in the posterior dorsum of the tongue. The posterior pharynx was also affected by a focus of carcinoma in case B4. Extension of the oesophageal carcinoma to involve surrounding tissues had occurred in two animals (B3, B11) with the development of a large carcinomatous mass in the subcutaneous tissues of the neck and ulceration of the skin in the former and infiltration of the tracheal wall without actual penetration into the lumen in the latter.

Metastasis was evident in two animals. Only the submandibular lymph nodes were involved in case B5 but in case B3 the carcinoma had disseminated widely and there were metastases present in the sternal and bronchial lymph nodes, the lungs, heart and one kidney.

Other discrete primary foci of upper alimentary squamous cell carcinoma were evident in eight animals (73%). The majority of these foci comprised small (3cm diameter) fungating or ulcerative lesions, but in five animals (B1, B4, B5, B6, B8) they were so numerous in the oesophagus that they constituted a generalised 'field change'(Figure 17). One animal (B4) had an extremely large (ca. 40 cms diameter) carcinoma of the dorsal non-pigmented area of the rumen.

Upper alimentary papillomas were found in every animal. Eight animals had oropharyngeal papillomas and with the exception of case B10 all had papillomatosis of the oesophagus and/or rumen.

FIGURE 17 Squamous cell carcinoma of the oesphagus showing generalised "field change"

Examination of the lower alimentary tract revealed the presence of adenomas and adenomatous hyperplasia in the small intestines and colons of three animals (B6, B7, B9) and in case B8 three adenocarcinomas of the small intestine. A submucosal lipoma was present in the colon of case B10.

Urinary bladder neoplasia was identified in one animal (B4) several small haemangiomas being found. Other neoplasms which were detected were a phaeochromocytoma of the left adrenal gland (B8), an adenoma of the gall bladder (B9) and a skin melanoma on the right shoulder of the foetus found in case B10.

iii) Ruminal Tympany Syndrome

Case Histories

A history was available for 12 of the 13 animals. Progressive loss of condition over a period of one week to six months had been observed in all the animals. Subsequently tympany of the rumen had developed in nine of the animals accompanied in three by diarrhoea and, or cud-dropping. The loss of condition in the remaining animals was associated with chronic diarrhoea in two and cud-dropping in one. All 13 animals were female and aged between eight and 15 years. The individual case histories are summarised in Table 14.

Presenting Signs

Ruminal tympany and poor bodily condition were the main presenting clinical signs in every animal and profuse diarrhoea was an additional presenting sign in the majority (82%) of cases.

Clinical Signs

The major clinical findings for each animal are summarised in Table 15.

All the animals were in poor bodily condition and afebrile. The majority (69%) were bright.

Significant clinical signs were mainly referable to the alimentary tract. A variable degree of ruminal tympany was a feature in every animal and in most (85%) the tympany was only mild or moderate causing bulging of the left paralumbar fossa and slight ventral abdominal distension. However in two animals (C8, C9) the tympany was severe resulting in massive abdominal distension and post-admission

TABLE 14

Ruminal Tympany Syndrome

Summary of Case Histories

Case Number	Breed	Age	Loss of Condition	Ruminal Tympany	Other
C1	High X	11y	One month	Three weeks (Continuous, mild)	
C2	Sh X	13y	Two months		Cud dropping for ten days
C3	AA	12y	Several weeks		Persistent diarrhoea for several weeks
C4	High X	15y	Six months	Three weeks (Intermittent, mild)	Cud dropping for three weeks and diarrhoea for three days
C5	High	13y	Several months	One week (Continuous, mild)	
C6	AA	>8y	No history available		
C7	AA	10y	One week	Three days (Continuous, mild)	Persistent diarrhoea for one week
C8	Sh X	10y	Several weeks	Three days (Continuous, severe)	
C9	High X	10y	Three weeks	Two days (Continuous, severe)	
C10	Gall X	8y	Several months	Two weeks (Continuous, mild)	
C11	High X	9y	Three weeks	Three days (Continuous, mild)	
C12	AA X	14y	Six months		Intermittent diarrhoea for six months
C13	High X	9y	Two weeks	Three days (intermittent, mild)	Cud dropping for ten days

TABLE 15

Ruminal Tympany Syndrome

Summary of Major Clinical Findings

Case Number	Condition	Demeanour	Rumen Tympany	Diarrhoea	Oropharyngeal Papillomas	Resistance to Stomach Tube
C1	Poor	Dull	Mild	-	Few	-
C2	Poor	Bright	Mild	-	Solitary	Complete
C3	Poor	Bright	Moderate	Profuse	Numerous	-
C4	Poor	Bright	Moderate	Profuse	Few	Complete
C5	Poor	Dull	Mild	-	Few	Marked
C6	Poor	Bright	Mild	Profuse	Few	Slight
C7	Poor	Bright	Mild	Profuse	Numerous	Moderate
C8	Poor	Dull	Severe	Profuse	Numerous	Moderate
C9	Poor	Dull	Severe	Profuse	Few	Marked
C10	Poor	Bright	Mild	-	Few	-
C11	Poor	Bright	Moderate	Profuse	-	Complete
C12	Poor	Bright	Mild	Profuse	Numerous	-
C13	Poor	Bright	Mild	Profuse	Few	Marked

severe tympany developed in a further three (C2, C7, C12). Spontaneous remission of tympany occurred post-admission in two animals (C6, C12), but in the latter it rapidly redeveloped and eventually became severe. As a consequence of the tympany, rumen contractions were reduced in intensity and duration, and in the most severely affected animals, contractions were completely abolished. Passage of a tube into the rumen was attempted in all the animals, but only in four could this procedure be accomplished with ease. An obstruction in the distal intra-thoracic oesophagus caused resistance to passage of the tube in six animals (C5, C6, C7, C8, C9, C13) and completely prevented entry of the tube into the rumen in the remaining three (C2, C4, C11). Despite the release of rumen gases when passage of a stomach tube was possible, in every case the recurrence of ruminal tympany was rapid and necessitated frequent relief in the more severely affected animals.

Profuse diarrhoea was present in nine animals (69%) and in six (C4, C6, C7, C8, C9, C12) it was characterised by the presence of long (1-3 cm) fibres of undigested hay. One further case (C1) developed diarrhoea post-admission, and of the remaining animals, one (C2) had normal faeces, one (C5) passed only scanty amounts of tarry faeces, and the last (C10) did not pass any during the two days between its admission and death. Over half the animals had a reduced appetite and three (C8, C9, C10) were totally anorexic on admission.

As in the oropharyngeal and oesophageal syndromes, oropharyngeal papillomas were detected in a high proportion of cases (92%), but other clinical signs frequently encountered in these syndromes were uncommon. Halitosis was evident in three animals (C5, C6, C10), each of which also had a mucopurulent nasal discharge, in one case (C10) containing ingesta. Cud-dropping was observed in only two animals (C2, C11) as were gurgling fluid sounds from the oesophagus (C6, C13). None of the animals had a mass in the oropharynx or cervical oesophagus and dribbling of saliva was not observed except in one case (C2) post admission. Coughing was heard only in one animal (C11) and other abnormalities of the respiratory tract were equally rare.

Other significant clinical signs were confined to the cardiovascular and urinary systems. Pallor of the mucosae was evident in five animals (C1, C3, C6, C9, C13). Haematuria was observed in three animals on admission (C3, C5, C13) but there was rapid remission in one case (C13). However haematuria subsequently developed in one further animal (C6).

Haematology

The results of haematological examinations performed on individual animals on admission are recorded in Appendix 3. The mean values of packed cell volume,

haemoglobin concentration and erythrocyte count are at the lower extreme of the normal range (Table 16) but only three animals (C1, C3, C13) were anaemic (packed cell volume <25%, haemoglobin concentration <8g/100mls). The anaemia was normocytic and hypochromic in two of these animals (C1, C3) and normocytic and normochromic in the other (C13).

The mean total leukocyte count was within the normal range (Table 16). Two of the animals had marked leukopaenia (<3.5 x 10^3 leukocytes/mm^3) which, in both cases, was due to both neutropaenia and lymphopaenia.

Biochemistry

The results of blood biochemical analysis performed on each animal on admission are recorded in Appendix 3. Abnormal values of plasma albumin and globulin were found in a high proportion of the animals with the result that the mean value for plasma albumin was markedly depressed and the mean value for plasma globulin was markedly raised (Table 17). Plasma albumin was low (<25g/l) in every animal and was markedly depressed (<20g/l) in eight cases (62%). Plasma globulin was raised (>55g/l) in all the animals and was markedly elevated (>65g/l) in seven cases (54%).

In addition, the mean values of plasma calcium and magnesium were outwith the lower limits of the normal range (Table 17) and six and 11 of the twelve animals in which these parameters were measured respectively had low values. The mean values of the other plasma constituents

TABLE 16

Ruminal Tympany Syndrome

Summary of Haematological Parameters

on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	13	28.8 ± 5.2	27 - 35
Haemoglobin (g/100mls)	13	9.3 ± 2.1	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	13	5.17 ± 1.03	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	13	32.1 ± 2.9	30 - 36
Mean Cell Volume (μ^3)	13	56.2 ± 6.5	40 - 60
Leukocyte Count ($x10^3/mm^3$)	13	6.4 ± 2.5	7 - 10

TABLE 17

Ruminal Tympany Syndrome

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	13	7.6 ± 3.8	0 - 8.3
SODIUM mmol/l	13	141 ± 7	136 - 151
POTASSIUM mmol/l	13	4.1 ± 0.7	3.2 - 5.8
CHLORIDE mmol/l	13	100 ± 6	96 - 111
CALCIUM mmol/l	12	2.22 ± 0.23	2.29 - 3.08
MAGNESIUM mmol/l	12	0.52 ± 0.10	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	13	2.02 ± 0.64	1.13 - 2.84
BILIRUBIN µmol/l	13	6 ± 6	0 - 8
ALKALINE PHOSPHATASE IU/l	13	75 ± 42	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	13	144 ± 94	0 - 200
ALANINE AMINOTRANSFERASE IU/l	13	35 ± 18	0 - 40
TOTAL PROTEIN g/l	13	87 ± 10	50 - 90
ALBUMIN g/l	13	18 ± 4	25 - 40
GLOBULIN g/l	13	69 ± 10	25 - 55

which were measured were within their normal ranges (Table 17) and in individual animals markedly abnormal values were infrequently encountered.

Faecal Examination

On admission, the faeces of all nine diarrhoeic animals were examined for the presence of nematode eggs and acid fast bacilli resembling M.paratuberculosis. The results for each individual animal are recorded in Appendix 3.

Trichostrongyle eggs were identified in five animals (C3, C4, C8, C9, C13) which had counts ranging between 50 and 400 epg of faeces. The results of faecal examination for the presence of acid fast bacilli resembling M.paratuberculosis were negative in all nine animals.

Serum Pepsinogin Estimation

Serum pepsinogen estimations were performed on admission for all nine diarrhoeic animals (Appendix 3). In eight animals the serum pepsinogen was less than 2000 mU tyrosine and in the remaining animal (C4) the value was 2794 mU tyrosine. In this animal, the serum pepsinogen level had fallen to 1965 mU tyrosine within one week of admission but diarrhoea persisted.

Urine Examination

Urine from each animal was examined on admission for the presence of protein and erythrocytes (Appendix 3. The urine protein was raised (>50mg/100ml) in four animals

(C2, C3, C5, C13) of which one (C3) had marked proteinuria. Microscopic examination revealed the presence of erythrocytes in three animals (C3, C5, C13) all of which had clinical haematuria and raised urine protein levels.

Pathological Findings

The major focus of carcinoma was situated at the cardia in nine animals, the distal extremity of the oesophagus in two (C11, C13) and on the dorsal non-pigmented area of the rumen in the remaining two (C4, C9). Extension of the major lesion by local infiltration to involve other organs had occurred in only one animal (C4) in which there was massive peritoneal spread of the carcinoma.

Metastasis was evident in ten animals (77%) and in all but one (C9) the mediastinal lymph nodes were involved. Other organs in which metastases were found were the liver (C6, C9) hepatic lymph nodes (C8) pleurae (C4) and lungs (C13).

Other discrete primary foci of upper alimentary squamous cell carcinoma were found in five animals (38%) situated in the oesophagus (C6, C7), cardia (C5) and rumen (C1, C5, C12). The majority of these foci comprised small (<3cms diameter) ulcerative lesions, but in two animals (C5, C6) slightly larger carcinomas were present in the rumen and oesophagus respectively.

Upper alimentary papillomas were found in every animal. All the animals had oropharyngeal papillomas and,

with the exception of cases C2, C3 and C13, papillomatosis of the oesophagus and, or rumen.

Examination of the lower alimentary tract revealed the presence of adenomas and adenomatous hyperplasia in the small intestines of seven animals (C1, C3, C4, C6, C10, C11, C12). In one animal (C3) two foci of adenocarcinoma were found in the caecum and colon and these had metastasised to the serosae of the abdominal organs, the mediastinum and the interlobular septae of the lungs. A submucosal lipoma was present in the colon of case C5.

Urinary bladder neoplasia was identified in two animals comprising three protruding haemangiosarcomas with metastases to the colic lymph nodes (C3) (Figure 18) and a haemangioma (C6). Other neoplasms which were detected were a phaeochromocytoma of an adrenal gland (C3), a renal cortical adenoma (C9), a fibroma of the oesophagus (C4) and a lipoma of the colon (C5).

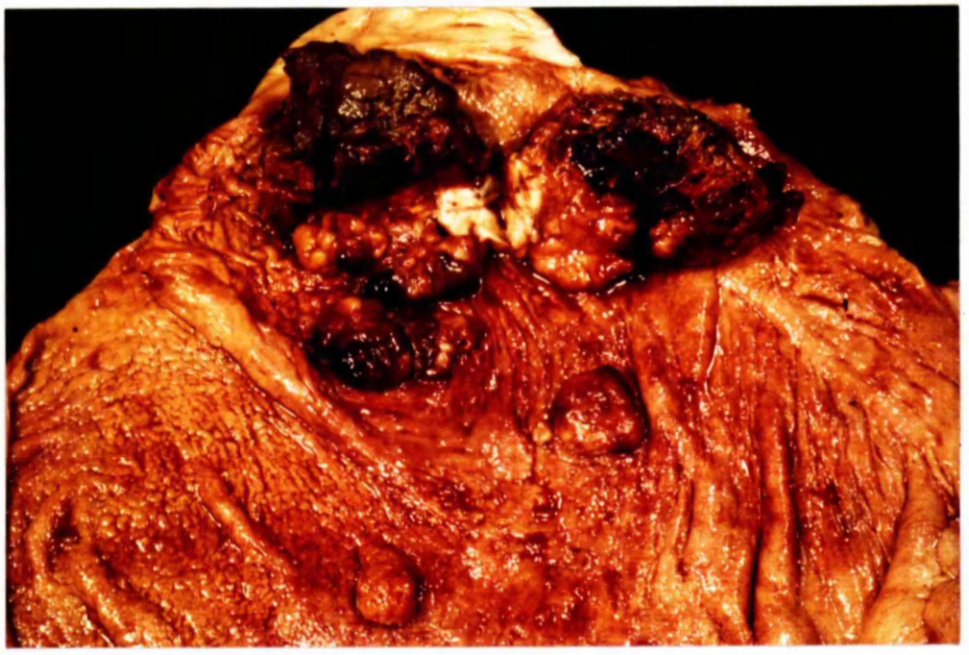


FIGURE 18 Haemangiosarcomas of the urinary bladder

(iv) Wasting and Diarrhoea Syndrome

Case Histories

A history was available for 11 of the 12 animals and in every case there had been loss of condition in association with chronic diarrhoea, over periods of several weeks to nine months. In three animals the loss of condition was noticed prior to the onset of diarrhoea but in the majority both were observed simultaneously. Subsequently the diarrhoea was intermittent in half the animals. During the week prior to admission mild ruminal tympany was observed in one animal and haematuria in another. All 12 animals were female and were aged between eight and 17 years. The individual case histories are summarised in Table 18.

Presenting Signs

Poor bodily condition and profuse diarrhoea were the presenting signs in every animal.

Clinical Signs

The major clinical findings for each individual animal are summarised in Table 19.

All the animals were in poor condition and afebrile. The majority (75%) were of bright demeanour.

Significant clinical signs were mainly referable to the alimentary tract. Profuse diarrhoea was a constant feature and in five animals (D1, D7, D10, D11, D12) it was characterised by the presence of long (1-3 cms) fibres of undigested hay (Figures 19 & 20). However the abdominal volume of the animals was varied, being reduced in six (50%), whereas

TABLE 18

Wasting/Diarrhoea Syndrome

Summary of Case Histories

Case Number	Breed	Age	Loss of Condition	Diarrhoea	Other
D1	High	>10y		No history available	
D2	Gall	>10y	Several months	Several months (Intermittent)	
D3	Gall X	10y	Six weeks	One week (Persistent, profuse)	
D4	AA	12y	Several weeks	Several weeks (Intermittent,profuse)	
D5	AA	13y	Nine months	Nine months (Intermittent,profuse)	
D6	Sh X	12y	One month	One month (Persistent)	
D7	Sh X	17y	Several weeks	Several weeks (Persistent)	Ruminal tympany observed three days prior to admission
D8	High X	9y	Six weeks	Six weeks (Persistent)	
D9	AA X	11y	Two months	Two months (Intermittent,profuse)	
D10	Sh X	8y	Several weeks	Several weeks (Persistent)	
D11	AA X	10y	Two months	Six weeks (Persistent)	
D12	Her X	14y	Three months	Two months (Persistent)	Haematuria during week prior to admission

TABLE 19

Wasting/Diarrhoea Syndrome

Summary of Major Clinical Findings

Case Number	Body Condition	Demeanour	Diarrhoea	Abdomen Size	Oropharyngeal Papillomas	Pallor of Mucosae
D1	Very poor	Bright	Profuse	Reduced	Numerous	Moderate
D2	Poor	Dull	Profuse	Normal	Solitary	Marked
D3	Poor	Dull	Profuse	Reduced	Solitary	-
D4	Poor	Bright	Profuse	Normal	Numerous	Marked
D5	Very poor	Bright	Profuse	Distended	Numerous	Marked
D6	Poor	Bright	Profuse	Reduced	Numerous	-
D7	Very poor	Bright	Profuse	Distended	Solitary	Slight
D8	Very poor	Bright	Profuse	Reduced	Numerous	-
D9	Poor	Dull	Profuse	Reduced	Numerous	Moderate
D10	Poor	Bright	Profuse	Reduced	Few	-
D11	Poor	Bright	Profuse	Distended	Numerous	-
D12	Poor	Bright	Profuse	Distended	Numerous	Slight

FIGURE 19 Diarrhoeic faeces showing long fibres of undigested hay suspended in fluid phase

FIGURE 20 Long fibres of undigested hay evident with draining of fluid phase shown in Figure 19

in four (33%) there was bilateral ventral distension. Although those with abdominal distension showed no evidence of tympany on admission, mild ruminal tympany did develop in one (D7) subsequently. On auscultation the majority of the animals (66%) had strong, regular ruminal contractions, and only two (D2, D3) were anorexic, both of which were found to have palpably enlarged livers, extending 10 and 12 cms respectively beyond the margins of the costal arch.

Examination of the oropharynx revealed the presence of papillomas in every animal. In addition, one animal (D5) was found to have a deep excavation in the posterior body of the tongue, this being the only case in which halitosis was evident. Dribbling of saliva was equally uncommon, and was only observed in one animal (D12) which had a large, traumatic ulcer of the lower lip. Four animals (D1, D7, D9, D11) had markedly worn teeth and in each case an incisor tooth was missing.

Respiratory signs were uncommon and the only abnormalities detected were occasional coughing in one animal (D2) and tachypnoea and, on auscultation, harsh respiratory sounds in two animals (D9, D11). Other significant clinical findings were confined to the cardiovascular and urinary systems. Pallor of the mucosae was evident in seven animals (58%), one of which (D2) had haematuria, and another (D12) a history of haematuria. One animal (D7) developed marked ventral abdominal oedema post admission, but apart from mucosal pallor no further cardiovascular abnormalities could be detected.

Haematology

The results of haematological examinations performed on individual animals on admission are recorded in Appendix 3. The mean values of packed cell volume, haemoglobin concentration and erythrocyte count are outwith the lower limit of the normal range (Table 20) and six animals (D1, D2, D4, D5, D9, D12) were anaemic (packed cell volume <25%, haemoglobin concentration <8g/100ml). The anaemia was normocytic and normochromic in three animals (D1, D4, D5) normocytic and hypochromic in two animals (D2, D12) and macrocytic and normochromic in the remaining animal (D9).

The mean total leukocyte count was within the normal range (Table 20). One animal had marked leukocytosis (>13.0 x $10^3/mm^3$) which was the result of neutrophilia.

Biochemistry

The results of blood biochemical analysis performed on each animal on admission are recorded in Appendix 3. Abnormal values of plasma albumin and globulin were found in a high proportion of the animals with the result that the mean value for plasma albumin was markedly depressed and the mean value for plasma globulin was markedly raised (Table 21). Plasma albumin was low (<25g/l) in all but one of the animals (D11) and was markedly depressed (<20g/l) in eight cases (67%). Plasma globulin was raised (>55g/l) in all but one of the animals (D2) and was markedly elevated (> 65g/l) in seven cases (58%).

TABLE 20

Wasting/Diarrhoea Syndrome

Summary of Haematological Parameters

on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	12	25.0 ± 5.6	27 - 35
Haemoglobin (g/100mls)	12	8.0 ± 2.1	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	12	4.54 ± 1.06	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	12	31.8 ± 2.8	30 - 36
Mean Cell Volume (μ^3)	12	55.3 ± 5.6	40 - 60
Leukocyte Count ($x10^3/mm^3$)	12	7.3 ± 3.5	7 - 10

TABLE 21

Wasting/Diarrhoea Syndrome

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	12	5.4 ± 2.2	0 - 8.3
SODIUM mmol/l	12	139 ± 7	136 - 151
POTASSIUM mmol/l	12	3.8 ± 1.0	3.2 - 5.8
CHLORIDE mmol/l	12	102 ± 6	96 - 111
CALCIUM mmol/l	12	2.40 ± 0.44	2.29 - 3.08
MAGNESIUM mmol/l	12	0.56 ± 0.19	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	12	1.29 ± 0.30	1.13 - 2.84
BILIRUBIN µmol/l	12	5 ± 4	0 - 8
ALKALINE PHOSPHATASE IU/l	12	63 ± 34	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	11	144 ± 83	0 - 200
ALANINE AMINOTRANSFERASE IU/l	11	33 ± 22	0 - 40
TOTAL PROTEIN g/l	12	84 ± 9	50 - 90
ALBUMIN g/l	12	17 ± 6	25 - 40
GLOBULIN g/l	12	67 ± 9	25 - 55

In addition, the mean value of plasma magnesium was outwith the lower limit of the normal range (Table 21) and six of the animals had low values. The mean values of the other plasma constituents which were measured were within their normal ranges (Table 21) and in individual animals markedly abnormal values were infrequently encountered.

Faecal Examination

On admission, the faeces of all the animals were examined for the presence of nematode eggs and acid fast bacilli resembling M. paratuberculosis. The results for each individual animal are recorded in Appendix .

Trichostrongyle eggs were identified in three animals (D1, D3, D5) which had counts of 50, 1150 and 200 epg of faeces respectively. The results of faecal examination for the presence of acid fast bacilli resembling M. paratuberculosis were negative in all the animals with one exception (D6) which was found to be positive. Repeat samples for this case were inconclusive and negative and repeat samples in all other cases were negative.

Serum Pepsinogen Estimation

Serum pepsinogen estimations were performed on admission in all the animals (Appendix 3). In ten animals the serum pepsinogen was less than 2000 mU tyrosine and in the remaining two (C6, C10) the values were 2714 and 2430 mU tyrosine respectively. In these two latter animals the

serum pepsinogen levels had fallen to 1983 and 939 mU tyrosine respectively, two and four weeks post admission, but diarrhoea persisted.

Urine Examination

Urine from each animal was examined on admission for the presence of protein and erythrocytes (Appendix 3). The urine protein was raised (>50mg/100ml) in six animals (D2, D3, D6, D8, D9, D12) but in none of these was marked proteinuria present. Microscopic examination revealed the presence of erythrocytes in the one animal (D2) which was clinically haematuric.

Pathological Findings

The rumen was the site of the major foci of carcinoma in every case. The main lesion was situated on the dorsal non-pigmented area of the rumen in eight animals and was composed of a single primary focus of carcinoma in four (D1, D4, D10, D11), and multiple primary foci in the remainder (D5, D6, D7, D9). The lesion also involved the cardia and terminal oesophagus to a major extent in one animal (D7) and there was an additional large primary focus of carcinoma involving the anterior ruminal pillar in another (D5). In the remaining four animals the sites involved were the anterior ruminal pillar (D2) the left lateral wall (D3), the dorsal margin of the oesophageal groove (D8) and the cardia and terminal oesophagus (D12). Infiltration of carcinoma through the ruminal wall to involve other structures in the abdominal cavity had occurred in five animals (D2, D3, D7, D8, D9).

Metastasis was evident in seven animals (58%). The liver was affected in five animals (D1, D2, D3, D9, D12) and in three of these (D2, D3, D9) infiltration was extensive. Other sites of metastases were the mediastinal lymph nodes (D2, D3, D5, D7, D9, D12) various other lymph nodes (D3, D5, D12), lungs (D7, D9), pleura (D3), diaphragm (D3) and duodenal lymphatics (D9).

Other discrete primary foci of upper alimentary squamous cell carcinoma were evident in nine animals (75%), most commonly situated in the oesophagus. The majority of these foci comprised small (<3cms diameter) fungating or ulcerative lesions. In case D5 a large excavating squamous cell carcinoma was present in the left posterior body of the tongue and there was generalised "field change" in the oesophagus.

Upper alimentary papillomas were found in every animal. All the animals had oropharyngeal papillomas and with the exception of case D4, all had papillomatosis of the oesophagus and/or rumen.

Examination of the lower alimentary tract revealed the presence of adenomas and adenomatous hyperplasia in the small intestines of eight animals (D2, D4, D5, D6, D7, D10, D12). Despite the initial identification of acid fast bacilli resembling M.paratuberculosis in case D6, there was no evidence of Johnes disease on examination of the intestinal mucosa.

Urinary bladder neoplasia was identified in three animals comprising multiple haemangiomas (D2) transitional cell carcinoma (D4) and a fibroma (D5). Other neoplasms which were detected were a fibroleimyoma of the uterus (D7), a phaeochromocytoma of the left adrenal (D7), a rumen fibroma (D8), teat papillomas (D9) and an adenoma of the gall bladder (D6).

(v) Atypical Clinical Cases of Upper Alimentary Squamous Cell Carcinoma

Three animals exhibited clinical signs dissimilar to those of the four main clinical syndromes and are thus described individually.

Case Number F1

Case History

This 16 year old Highland cow had shown slow progressive loss of condition over a period of approximately one year.

Clinical Signs

The animal was in very poor body condition, but was bright and afebrile. The only significant clinical findings were pallor of the oral conjunctival and vulval mucosae and the presence of numerous papillomas on the tongue and walls of the pharynx. Throughout the first month post admission, there was no change in the clinical condition of the animal but, during the following month, pallor of the mucosae became increasingly marked and the animal progressively weaker. Terminally severe respiratory signs developed with tachypnoea, hyperpnoea, frequent spontaneous coughing, a mucopurulent nasal discharge and, on auscultation, rhonchi in the anteroventral areas of the lung fields.

Haematology

On admission, haematological examination demonstrated the presence of a macrocytic, normochromic anaemia (Table 22) but there was no evidence of reticulocytosis (reticulocyte

TABLE 22

Atypical Clinical Forms of Upper Alimentary Squamous Cell Carcinoma

Haematological Parameters on Admission

PARAMETER	CASE NUMBER F1	CASE NUMBER F2	CASE NUMBER F3	NORMAL RANGE
Packed Cell Volume (%)	22	28	39	27 - 35
Haemoglobin (g/100mls)	7.9	-	12.5	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	3.20	-	6.81	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	35.9	-	32.1	30 - 36
Mean Cell Volume (μ^3)	69	-	57	40 - 60
Leukocyte Count ($x10^3/mm^3$)	2.1	4.0	5.8	7 - 10

count <1%). Post admission the anaemia became more marked and in two months the anaemia was extremely severe (packed cell volume, 12%, haemoglobin concentration 1.58g/100mls, erythrocyte count 4.4 x $10^6/mm^3$). The anaemia continued to be macrocytic and normochromic and the reticulocyte count never exceeded one per cent.

There was also a marked leukopaenia on admission which was the result of both neutropaenia and lymphopaenia. The leukopaenia also became more severe and by two months post admission the total leukocyte count was 1.3 x $10^3/mm^3$ (14% neutrophils, 1% eosinophils,85% lymphocytes).

Biochemistry

The results of blood biochemical analysis performed on admission are recorded in Table 23. The only plasma constituent which was found to be outwith the normal range was plasma globulin which was markedly elevated. Plasma albumin was at the extreme lower end, although not outwith, the normal range.

Urine Examination

The urine protein was not raised and no erythrocytes were found on microscopic examination..

Pathological Findings

Numerous, shallow, irregular ulcers which ranged in size between three millimetres and two centimetres in diameter were scattered throughout the length of the

TABLE 23

Atypical Clinical Forms of Upper Alimentary Squamous Cell Carcinoma

Biochemical Parameters on Admission

PLASMA CONSTITUENT	CASE NUMBER F1	CASE NUMBER F2	CASE NUMBER F3	NORMAL VALUES
UREA mmol/l	5.8	3.8	4.1	0 - 8.3
SODIUM mmol/l	145	148	153	136 - 151
POTASSIUM mmol/l	4.6	4.5	3.6	3.2 - 5.8
CHLORIDE mmol/l	109	107	108	96 - 111
CALCIUM mmol/l	2.72	2.35	2.38	2.29 - 3.08
MAGNESIUM mmol/l	0.78	0.53	0.82	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	2.03	1.45	0.97	1.13 - 2.84
BILIRUBIN µmol/l	2	2	1	0 - 8
ALKALINE PHOSPHATASE IU/l	57	36	34	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	104	153	92	0 - 200
ALANINE AMINOTRANSFERASE IU/l	21	23	31	0 - 40
TOTAL PROTEIN g/l	102	92	100	50 - 90
ALBUMIN g/l	25	13	26	25 - 40
GLOBULIN g/l	77	79	74	25 - 55

oesophagus. Microscopic examination confirmed that the ulcers comprised squamous cell carcinoma. Numerous papillomas were associated with the ulcers and together they constituted a generalised field change. Papillomas were also present on the tongue and in the pharynx. The femoral and sternal bone marrow was yellow gelatinous and non-reactive. In addition, a severe purulent bronchopneumonia affected the apical, cardiac and anterior diaphragmatic lung lobes.

Case Number F2

Case History

This was an Aberdeen Angus cow, aged in excess of ten years, for which no history was available.

Clinical Signs

The animal was in very poor body condition, dull and afebrile. Slight tachypnoea and hyperpnoea were evident and occasionally the cow was heard to cough. On auscultation of the lungs rhonchi were audible in the diaphragmatic area of the right lung field. In addition, reduced resonance and pain were appreciable on percussion of this area and extending over the area of the liver. The liver was palpably enlarged and extended 1.5 inches beyond the margin of the right dorsal costal arch. There was no diarrhoea or ruminal tympany and the ruminal contractions were strong and regular. Two small papillomas were detected on the smooth part of the hard palate.

Haematology

The only red cell parameter which was measured was the packed cell volume which was within the normal range (Table 22). The total leukocyte count was depressed ($4.0 \times 10^3/mm^3$) but, unfortunately, no differential count was performed.

Biochemistry

The results of blood biochemical analysis performed on admission are recorded in Table 23. Abnormal values were found for plasma albumin and globulin which were markedly depressed and elevated respectively. In addition plasma magnesium was slightly depressed.

Urine Examination

A marked proteinuria (280mg/100mls) was detected on admission but no erythrocytes or other deposit were found on microscopic examination.

Pathological Findings

The main lesion was a large (30 x 24 x 15cm) squamous cell carcinoma which involved the dorsal non-pigmented area of the rumen and had, by direct extension, penetrated the ruminal wall, eroded through the diaphragm and infiltrated the lungs and mediastinal lymph nodes. A second focus of carcinoma measuring seven centimetres in diameter was present in the ventral sac of the rumen and the adjacent, anterior rumenal pillar was heavily infiltrated. Numerous metastases were scattered throughout the liver producing marked hepatic enlargement. The hepatic lymph nodes were also enlarged

and were almost completely replaced by tumour tissue. Two small papillomas were identified on the smooth part of the hard palate, and a few small areas of adenomatous hyperplasia were noted in the duodenum and colon.

Case Number F3

Case History

This was a ten year old Aberdeen Angus cross cow in which a mass involving the anterior mandible had been observed one week prior to admission.

Clinical Signs

The cow was in good body condition, bright and afebrile. A lobulated, ulcerated mass involving the lower gum and lip was situated to the left of the central incisor teeth (Figure 21). There was marked halitosis. No other significant clinical signs were detected.

Haematology

There was no evidence of anaemia (Table 22). The total leukocyte count was marginally outwith the lower limits of the normal range.

Biochemistry

The results of blood biochemical analysis performed on admission are recorded in Table 23. The only significant abnormality was a markedly elevated plasma globulin level.

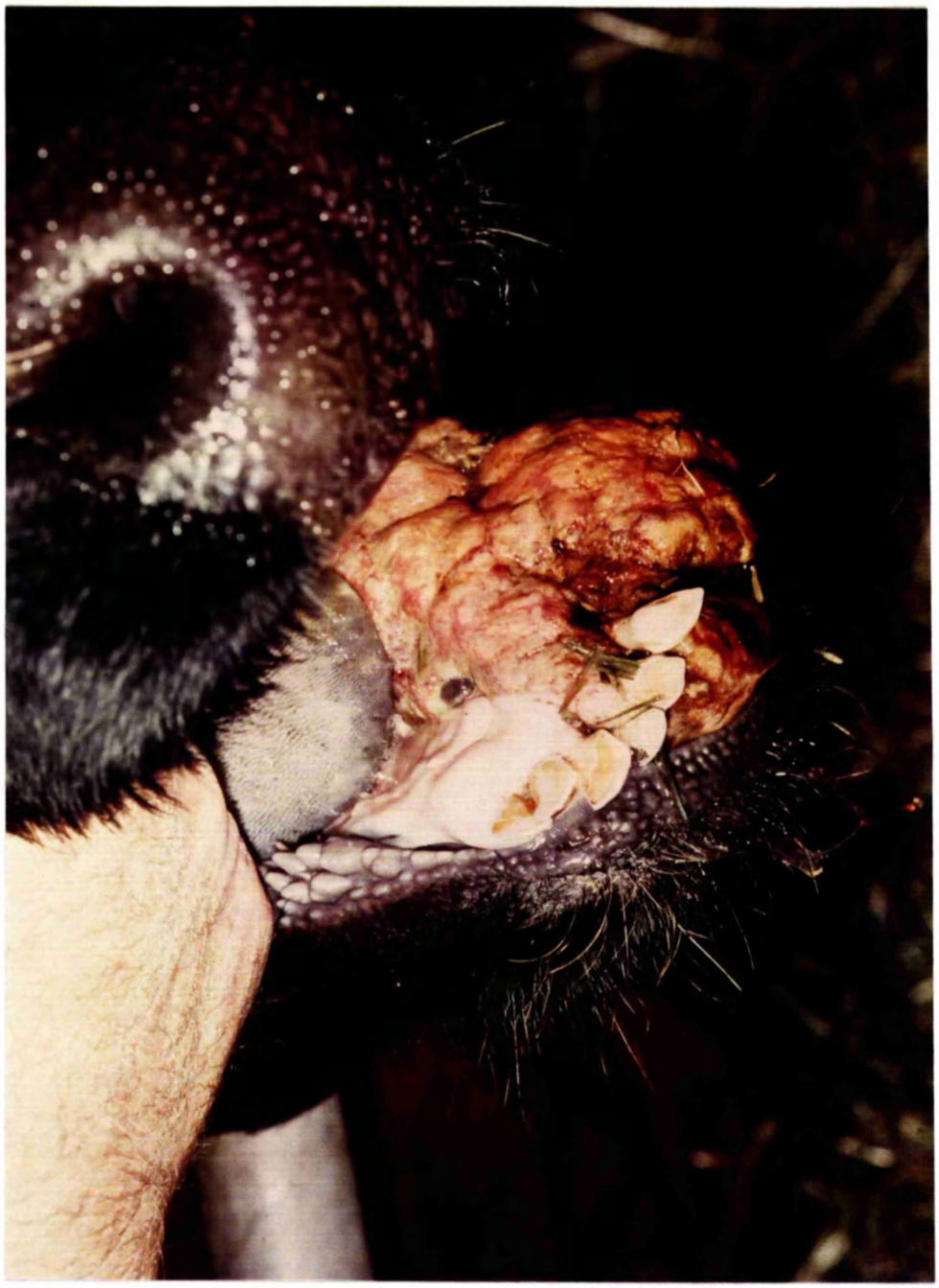


FIGURE 21 Squamous cell carcinoma of the mucosa of the gum and lower lip

Urine Examination

No protein was detected in the urine and no erythrocytes were found on microscopic examination.

Pathological Findings

The mass involving the lower gum and lip was found to be a squamous cell carcinoma. No metastases were detected. Papillomas were present in the pharynx and on the root of the tongue.

DISCUSSION

During the course of the study it was found that with careful assessment of the clinical signs observed in individual animals it was possible to reach an accurate clinical diagnosis in a high proportion of cases.

The clinical signs observed in animals affected by upper alimentary squamous cell carcinoma could, in most cases, be directly attributed to the physical effects of the tumour mass(es) identified at post mortem examination.

Four clinical syndromes were recognised depending on whether the neoplasm was situated in the oropharynx, the oesophagus, the distal oesophagus or cardia, or the rumen. However, in a number of animals the main tumour mass was situated at an interface between these organs or there was more than one clinically significant tumour mass. In such cases clinical signs typical of more than one of the four syndromes could be observed. The most striking example of this situation is case D7 which, on examination at admission, had clinical signs typical of the wasting and diarrhoea syndrome but both prior to the post-admission exhibited ruminal tympany. However, on pathological examination the changing clinical picture is readily explained by the presence of carcinoma, not only involving the wall of the rumen, as in other cases which exhibited the wasting and diarrhoea syndrome, but also the terminal oesophagus and cardia as was usual in animals with the ruminal tympany syndrome.

The few cases which presented any difficulty in clinical diagnosis were those in which loss of condition and profuse diarrhoea were the only significant clinical signs. In such animals it was necessary to exclude other clinically similar conditions, principally Johnes disease and ostertagiasis. This was effected by the use of simple laboratory techniques comprising examination of faeces for the presence of acid fast bacilli resembling M.paratuberculosis in the case of Johnes disease, and measurement of serum pepsinogen levels and faecal egg counts in the case of ostertagiasis.

The main haematological and biochemical abnormalities identified in animals affected by upper alimentary squamous cell carcinoma were anaemia, hypoalbuminaemia and hyperglobulinaemia. Anaemia, which was a feature in 31 per cent of the animals, could be related to a number of causes. Secondary anaemia is commonly observed in human patients with cancer and appears to be primarily due to an increased rate of erythrocytic destruction (Kremer and Laszlo, 1973). This type of anaemia is usually mild or moderate, and normocytic and normochromic or normocytic and hypochromic, as in the majority of the animals affected by upper alimentary squamous cell carcinoma. Haemorrhage may also cause anaemia in human patients with cancer of the upper alimentary tract and occasionally anaemia is the presenting clinical sign in such patients, as in case F1. In addition, urinary bladder neoplasia was also present in some of the animals with upper alimentary sugmous cell carcinoma. Haematuria was evident in two of these cases (C3, D2) which were anaemic and it would

appear likely that this haemorrage would contribute to the development of anaemia.

Hypoalbuminaemia and hyperglobulinaemia were evident in 89 per cent and 93 per cent of animals with upper alimentary squamous cell carcinoma respectively. Factors likely to be of significance in the development of hypoalbuminaemia in these animals were decreased protein uptake due to interference with normal digestive function, decreased protein synthesis in cases with extensive tumour infiltration of the liver and, latterly, inanition. The presence of hyperglobulinaemia in a high proportion of cases may be analogous to the situation observed in human patients with cancer, in which there is often a non specific increase in the concentration of plasma globulins in addition to hypoalbuminaemia (Reynoso, 1973).

Pathological examination of the upper alimentary tract confirmed the relationship between the clinical signs observed and the location of foci of carcinoma. Usually the clinical signs in an individual animal could be attributed to the physical effects a single focus of carcinoma, despite the presence, in many cases, of multiple primary foci of carcinoma scattered throughout the upper alimentary tract. Diarrhoea was the only major clinical sign present in a high proportion of animals with all four syndromes which could not be directly attributed to the physical effects of foci of carcinoma. However there was no evidence of an association between diarrhoea and the presence of adenomas and, or

adomenatous hyperplasia of the intestines. The development of diarrhoea presumably occurred as a result of changes in ruminal function in those cases with large foci of carcinoma in the rumen and interference with the processes of rumination in those with foci of carcinoma in the orapharynx and oesophagus. Metastases were seldom responsible for obvious clinical abnormalities except in the few cases in which there was extensive metastatic infiltration of organs such as the liver,as in cases D2 and D3.

Previously, two clinical syndromes, one associated with carcinoma of the orapharynx or proximal oesophagus and the other with carcinoma of the rumen or distal oesophagus, have been described in broad terms by Dobereiner and others (1967), Plowright and others (1971) and Campos Neto and others (1975). However these studies only provide a general indication of the spectrum of clinical signs which can be observed in cattle affected by upper alimentary squamous cell carcinoma whereas the present study provides a comprehensive description of the clinico-pathological aspects of this condition.

SECTION II

CLINICAL ASPECTS OF URINARY BLADDER NEOPLASIA

RESULTS

Clinical examination of animals which were subsequently confirmed to be affected by urinary bladder neoplasia revealed that the animals exhibited a syndrome which was characterised clinically by haematuria.

Case Histories

A history was available for all 27 animals, and with only one exception the animals had been observed passing blood stained urine for a period ranging between two days and two years. In 15 cases the haematuria had been persistent from the time it was first observed but in the remaining 11 cases the appearance of blood in their urine had been intermittent, with remissions lasting for several days or, occasionally, weeks. Three animals, including the only case in which haematuria was not reported, had exhibited straining. Loss of condition was associated with the haematuria in four animals and diarrhoea had developed a few days prior to admission in two animals. All the animals were female and were aged between three and 16 years. The individual case histories are summarised in Table 24.

Presenting Signs

Haematuria was the main presenting sign in 22 animals (81%). In four of these cases straining (U2, U27) or profuse diarrhoea (U3, U24) were additional presenting signs. Three of the non-haematuric animals presented with poor body condition (U1, U6) or straining (U23) but in the

TABLE 24

Urinary Bladder Neoplasia

Summary of Case Histories

Case Number	Breed	Age	Haematuria		Other
			Duration	Occurrence	
U1	AAX	11yr	Several months	Intermittent	
U2	AAX	6yr	One week	Persistent	Straining for two days
U3	AAX	5yr	Two weeks	Persistent	
U4	Ayr	10yr	Several months	Intermittent	
U5	Ayr	5yr	Several months	Intermittent	
U6	High X	8yr	Two weeks	Persistent	Loss of condition
U7	AA	8yr	Three days	Persistent	
U8	AA	10yr	Three weeks	Persistent	
U9	Ayr	10yr	Three weeks	Persistent	
U10	AAX	12yr	One week	Persistent	
U11	Her X	3yr	One month	Intermittent	
U12	Ayr	7yr	Two weeks	Persistent	
U13	High	12yr	One month	Intermittent	Loss of condition
U14	Ayr	10yr	Three weeks	Persistent	

TABLE 24 (continued)

Urinary Bladder Neoplasia

Summary of Case Histories

Case Number	Breed	Age	Haematuria		Other
			Duration	Occurrence	
U15	Her X	11yr	Several months	Intermittent	Loss of condition
U16	Fr	12yr	Two years	Intermittent	
U17	AA	>6yr	Two weeks	Persistent	
U18	Ayr	9yr	One week	Persistent	Loss of condition
U19	Sh X	8yr	One week	Persistent	
U20	AA	16yr	Several weeks	Persistent	
U21	AA	12yr	Four months	Intermittent	
U22	Sh X	8yr	Three months	Intermittent	
U23	AA X	10yr	-	Never observed	Straining for 1 day
U24	Sh X	11yr	Three weeks	Persistent	Profuse diarrhoea for 3 days and straining for 1 day
U25	Sh X	9yr	Four days	Persistent	Occasional coughing for several weeks
U26	Gall X	12yr	Four months	Intermittent	Diarrhoea for three days
U27	Luing	8yr	Several	Intermittent	

remaining two cases (U5, U19) there were no obvious abnormalities on initial visual examination.

Clinical Signs

The major clinical findings for each individual animal are summarised in Table 25.

The majority (67%) of the animals were in either moderate or good condition and with only two exceptions (U11, U14) were of bright demeanour. Two animals (U1, U24) were pyrexic.

Haematuria was evident in 23 animals (85%) but the severity was extremely variable. The spectrum of urine discolouration ranged from a reddish tinge and clouding in slightly haematuric animals, to a deep red coloration and opacity in those with marked haematuria. The degree of haematuria was considered slight in four animals, moderate in five and marked in 14. Clots of coagulated blood were present in the urine of five of the markedly haematuric animals (U8, U9, U10, U16, U22). (Figure 22).

An increase in the frequency of micturition was common in the haematuric animals but straining was restricted to four cases, three of which (U2, U23, U24) dribbled urine almost continuously. Dribbling of urine was a feature in a further two animals (U10, U13) which, although not straining, had abnormalities of the urinary tract detectable per rectum. Rectal and vaginal examinations revealed abnormalities of the urinary tract in nine animals (33%). These comprised a mass involving the bladder in five cases (U11, U13, U17, U23, U27), three of which also had enlarged left kidneys (U11, U23, U27),

TABLE 25

Urinary Bladder Neoplasia

Summary of Major Clinical Findings

Case No.	Body Condition	Demeanor	Clinical Haematuria	Pallor	Oropharyngeal Papillomas	Micturition		Urinary Abnormalities on Rectal Examination	
						Frequency	Straining	Bladder	Left Kidney
U1	Poor	Bright		Moderate					Enlarged
U2	Good	Bright	Slight			Increased	Frequent		
U3	Good	Bright	Marked	Marked	Few	Increased		Distention	
U4	Good	Bright	Slight		Numerous				
U5	Good	Bright			Few				
U6	Poor	Bright			Few				
U7	Moderate	Bright	Marked			Increased			
U8	Good	Bright	Marked	Moderate		Increased			
U9	Good	Bright	Marked	Marked	Numerous	Increased			
U10	Good	Bright	Marked	Marked		Increased		Distention	
U11	Good	Dull	Moderate	Slight		Increased		Mass	Enlarged
U12	Poor	Bright	Moderate						
U13	Poor	Bright	Moderate	Marked	Solitary	Increased		Mass	
U14	Moderate	Dull	Marked	Marked					

TABLE 25 (continued)

Urinary Bladder Neoplasia

Summary of Major Clinical Findings

Case No.	Condition	Demeanor	Clinical		Oropharyngeal Papillomas	Micturition		Urinary Abnormalities on Rectal Examination	
			Haematuria	Pallor		Frequency	Straining	Bladder	Left Kidney
U15	Poor	Bright	Marked	Moderate	Few	Increased			
U16	Moderate	Bright	Marked	Marked		Increased			
U17	Poor	Bright	Moderate		Numerous			Mass	
U18	Poor	Bright	Marked	Moderate	Numerous				
U19	Good	Bright							
U20	Good	Bright	Marked		Few	Increased			
U21	Good	Bright	Moderate		Numerous	Increased			
U22	Moderate	Bright	Marked	Marked	Few	Increased			
U23	Poor	Bright	Slight			Increased	Frequent	Mass	Enlarged
U24	Good	Bright	Marked	Marked		Increased	Occasional		
U25	Good	Bright	Slight						
U26	Moderate	Bright	Marked	Marked	Few	Increased		Distention	
U27	Poor	Bright	Marked	Moderate		Increased	Frequent	Mass	Enlarged

FIGURE 22 Urinary bladder neoplasia: Marked haematuria with the passage of clots of coagulated blood

massive distention of the bladder in three cases (U3, U10, U26) and enlargement of the left kidney in the absence of a detectable bladder mass in the remaining case (U10), which was the only non-haematuric animal exhibiting any clinical sign referable to the urinary tract.

Pallor of the mucosae was evident in 15 animals (56%), of which seven (U3, U9, U13, U15, U22, U24, U26) had tachycardia, three (U3, U24, U26) subcutaneous oedema and two (U3, U13) systolic cardiac murmurs.

The only significant clinical signs referable to the alimentary tract were the presence of oropharyngeal papillomas in 13 animals (48%) and diarrhoea in four animals (U3, U24, U26, U27).

Twelve animals (45%) were hyperpnoeic but in most cases this was not a marked feature. Other respiratory signs were uncommon; five animals (U6, U13, U19, U25, U27) were tachypnoeic, three (U6, U16, U25) coughed occasionally and in two (U6, U25), both of which were considered to have chronic pneumonias, adventitious sounds were detected on auscultation.

Urine Examination

The results of urine examination for the presence of erythrocytes, performed for each animal on admission, are recorded in Table 26. The presence of erythrocytes in the urine of the 23 animals which were clinically haematuric on admission was confirmed, and in addition, microscopic

TABLE 26

Urinary Bladder Neoplasia

Examination of Urine for the Presence of Erythrocytes

Case No.	Haematuria on admission		Constancy of Haematuria post-admission				Time from admission to slaughter/ death (days)
	Clinical	Microscopic	Clinical	Microscopic	High	Low	
U1	None	+	None	Intermittent	+	0	14
U2	Mild	++	Intermittent	Persistent	++++	+	30
U3	Marked	++++	Intermittent	Intermittent	++++	0	12
U4	Mild	++	Intermittent	Intermittent	+++	0	31
U5	None	0	None	None	0	0	33
U6	None	0	Intermittent	Intermittent	+++	0	17
U7	Marked	++++	Intermittent	Intermittent	++++	0	52
U8	Marked	+++++	Persistent	Persistent	+++++	++++	14
U9	Marked	+++++	Persistent	Persistent	+++++	+++	14
U10	Marked	+++++	Persistent	Persistent	++++	++	14
U11	Moderate	+++	Persistent	Persistent	+++	+++	6
U12	Moderate	+++	Intermittent	Intermittent	+++	0	77
U13	Moderate	+++	Persistent	-	-	-	<1
U14	Marked	++++	Persistent	-	-	-	1

TABLE 26 (continued)

Urinary Bladder Neoplasia

Examination of Urine for the Presence of Erythrocytes

Case No.	Haematuria on admission		Constancy of Haematuria post-admission				Time from admission to slaughter/ death (days)
	Clinical	Microscopic	Clinical	Microscopic	High	Low	
U15	Marked	++++	Persistent	-	-	-	2
U16	Marked	+++++	Intermittent	Intermittent	+++++	0	45
U17	Moderate	+++	Persistent	Persistent	+++++	+++	3
U18	Marked	++++	Persistent	Persistent	++++	+++	39
U19	None	+	None	Intermittent	+	0	14
U20	Marked	++++	Persistent	Persistent	+++++	++++	38
U21	Moderate	+++	Persistent	Persistent	+++++	+++	33
U22	Marked	+++++	Persistent	Persistent	+++++	++++	24
U23	Mild	++	Persistent	Persistent	++	++	15
U24	Marked	++++	Persistent	Persistent	++++	+++	2
U25	Mild	++	Intermittent	Persistent	++++	+	94
U26	Marked	++++	Persistent	-	-	-	1
U27	Marked	++++	Persistent	Persistent	+++++	++++	19

haematuria was evident in two animals (U1, U19) which were not clinically haematuric.

Twenty animals survived for more than one week post admission. Sixteen were clinically haematuric on admission and in nine of these haematuria was persistent. The remaining seven animals underwent temporary clinical remissions lasting between three and 60 days although two (U2, U25) had persistent microscopic haematuria. Four animals were not clinically haematuric on admission and of these one (U6) developed clinical haematuria post admission, two (U1, U19) had intermittent microscopical haematuria and one (U5) was never seen to be either clinically or microscopically haematuric. The major changes in degree of haematuria which occurred in each animal between admission and death or slaughter are recorded in Table 26.

On admission, urine was examined from each animal for the presence of protein. The amount of protein found was directly related to the degree of haematuria ($p = <0.001$) and ranged from 13 ± 9 mg/100mls in animals with no haematuria to 1416 ± 626 mg/100mls in those with marked haematuria (Table 27).

Haematology

The results of haematological examinations performed on individual animals are recorded in Appendix 3. The mean values of packed cell volume, haemoglobin concentration, erythrocyte count, mean cell haemoglobin concentration

TABLE 27

Urinary Bladder Neoplasia

Proteinuria Related to the Severity of Haematuria

Severity of Haematuria	0	+	++	+++	++++	+++++
Proteinuria (mg/100 mls) ± Standard Deviation	13 ± 9	84 ± 76	127 ± 174	573 ± 446	871 ± 472	1416 ± 626
Number of cases	2	2	4	5	9	5

(MCHC), mean cell volume (MCV) and leukocyte count are recorded in Table 28. The mean values of packed cell volume, haemoglobin concentration and erythrocyte count are outwith the lower limits of their normal ranges (Table 28). However there is a wide range in individual values for these parameters as is indicated by the large standard deviations.

Fourteen animals were anaemic (packed cell volume <25% and haemoglobin concentration <8g/100mls), all but one of which were moderately or markedly haematuric on admission, whereas haematuria was absent or only slight in seven of the 13 animals which were not anaemic. These differences are reflected in the mean values for packed cell volume, haemoglobin concentration and erythrocyte count of the moderately and markedly haematuric animals which are significantly lower ($p = <0.001$) than those of the animals in which haematuria was absent or only slight (Table 28).

The anaemia was normocytic and normochromic in the majority of the animals in which it was present, but in a few, particularly those with severe anaemia, it was macrocytic and normochromic (U3, U8, U14) or macrocytic and hypochromic (U22, U24, U26). This is reflected in the mean value for mean cell volume of the moderately and markedly haematuric animals which is outwith the upper limit of the normal range and is significantly greater ($p = <0.001$) than those of the animals with slight or no haematuria (Table 28).

TABLE 28

Urinary Bladder Neoplasia

Summary of Haematological Parameters on Admission

(Mean Values ± Standard Deviation)

PARAMETER	All Cases of Urinary Bladder Neoplasia	Animals with Marked or Moderate Clinical Haematuria	Animals with Slight or No Clinical Haematuria	Normal Range
Packed Cell Volume (%)	24.4 ± 9.1 (27)	20.8 ± 7.9 (19)	32.7 ± 6.0 (8)	27 - 35
Haemoglobin Concentration (g/100mls)	7.7 ± 3.1 (26)	6.7 ± 3.0 (19)	10.5 ± 1.8 (7)	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	4.10 ± 1.91 (26)	3.46 ± 1.63 (19)	5.85 ± 1.53 (7)	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	31.7 ± 3.8 (26)	31.2 ± 4.3 (19)	33.0 ± 1.6 (7)	30 - 36
Mean Cell Volume (μ^3)	62.4 ± 13.3 (26)	65.0 ± 14.4 (19)	55.3 ± 5.6 (7)	40 - 60
Leukocyte Count ($x10^3/mm^3$)	7.0 ± 3.4 (27)	5.8 ± 2.1 (19)	9.7 ± 4.4 (8)	6 - 10

The mean total leukocyte count of all the animals is within the normal range (Table 28). When only the animals with moderate or marked haematuria are considered, the mean value is slightly depressed. However marked leukocytosis (>13.0 x 10^3 leukocytes/mm^3) and marked leukopaenia (<3.5 leukocytes/mm^3) were each found in only one animal (U1 and U26 respectively).

Biochemistry

The results of blood biochemical analysis performed for each animal on admission are recorded in Appendix 3.

With the exception of albumin, the mean values of the affected animals were within the normal ranges for the plasma constituents measured (Table 29) and in individual animals markedly abnormal values were only occasionally encountered (Appendix 3).

Plasma albumin was low (<25 g/l) in 63 per cent of the animals, being markedly depressed (<20 g/l) in 33 per cent. However, hypoalbuminaemia was related to the degree of anaemia present, there being a highly significant correlation ($p = <0.001$) between the plasma albumin and the packed cell volume of the animals.

Faecal Examination and Serum Pepsinogen Estimation

Four animals were diarrhoeic (U3, U24, U26, U27) and faeces from each were examined for the presence of nematode eggs, and acid fast bacilli morphologically resembling Mycobacterium paratuberculosis. In every case the

TABLE 29

Urinary Bladder Neoplasia

Summary of Biochemical Parameters on Admission

Plasma Constituent	Number of Animals	Mean value ± Standard Deviation	Normal Range
Urea m mol/l	27	7.8 ± 11.9	0 - 8.3
Sodium m mol/l	27	141 ± 8	136 - 151
Potassium m mol/l	27	4.4 ± 1.1	3.2 - 5.8
Chloride m mol/l	27	100 ± 9	96 - 111
Calcium m mol/l	25	2.29 ± 0.34	2.29 - 3.08
Magnesium m mol/l	27	0.78 ± 0.23	0.65 - 1.39
Inorganic Phosphate m mol/m	27	1.86 ± 0.75	1.13 - 2.84
Bilirubin u mol/l	26	4 ± 3	0 - 8
Alkaline Phosphatase IU/l	27	63 ± 49	4 - 127
Aspartate Aminotransferase IU/l	27	135 ± 145	0 - 200
Alanine Aminotransferase IU/l	27	47 ± 55	0 - 40
Total Protein g/l	27	76 ± 17	50 - 90
Albumin g/l	27	23 ± 7	25 - 40
Globulin g/l	27	53 ± 14	25 - 55

examinations proved negative. Serum pepsinogen values for the four animals were 1460, 1520, 2192 and 857 mu tyrosine respectively.

Pathological Findings

Urinary bladder neoplasia was present in every animal, the types most frequently identified being haemangiomas and transitional cell carcinomas, one or both of which were found in all but two animals (U17, U27). Other urinary bladder neoplasms which were identified were haemangiosarcoma (U17) squamous cell carcinoma (U27) and fibroma (U18, U20). The number and size of neoplasms varied enormously from animal to animal. Where haemangiomas were present these tended to be multiple, ranging in size between one and 15 millimetres in diameter, but occasionally large solitary haemangiomas measuring up to six centimetres in diameter were found (U14, U15). Transitional cell carcinomas were usually solitary and ranged in size between two millimetres and 12 centimetres in diameter. In cases in which the carcinoma was large the bladder wall was thickened by tumour which extended into the muscular coats of the bladder. Occasionally perforation of the bladder wall had occurred resulting in extensive local adhesions between the bladder and surrounding organs (U13, U23). However, metastasis was rare and was only found in one animal (U17) in which a haemangiosarcoma had metastasised to the medial iliac lymph nodes.

The only common non-neoplastic lesion found in the urinary tract of the animals was cystitis which was present in 59 per cent of cases. In the majority the cystitis was mild with focal accumulations of lymphoid cells in the lamina propria and hyperplasia of the overlying epithelium, but, occasionally, the cystitis was severe (U3, U9) with diptherisis and necrosis of the bladder mucosa and massive infiltration of the submucosa by polymorphonuclear leukocytes. Renal lesions were uncommon. Three animals had chronic suppurative pyelonephritis (U1, U2, U23) and renal infarcts were present in two cases (U9, U11).

In addition to neoplastic lesions of the urinary bladder the majority of the animals had neoplasia of the alimentary tract. Nineteen animals (70%) had upper alimentary papillomas which were found in the oropharynx (15 animals), oesophagus (9 animals) and rumen (6 animals). Two animals (U7, U18) had small foci of squamous cell carcinoma in both the oesophagus and rumen, intestinal adenocarcinoma was present in two animals (U15, U18) and 11 animals had intestinal adenomatous plaques and polyps.

Other neoplastic lesions identified in four of the animals were thyroid adenomas (U10, U18), acidophil adenoma of the pituitary (U10), cortical adenoma of the adrenal (U20) and teat papillomas (U21).

DISCUSSION

The clinical findings in animals with urinary bladder neoplasia, which are described in this study, are similar to those which have been reported previously although earlier studies rarely contain detailed descriptions of the clinical signs in individual animals for which there is pathological confirmation of the diagnosis. The typical clinical picture was of a bright adult animal with intermittent or persistent haematuria which, in most cases, gradually increased in severity over a prolonged period of weeks or months. As in previous studies, other major clinical signs frequently observed were an increase in the frequency of micturition and, in markedly haematuric animals, pallor of the mucosae.

The palpation of abnormalities of the urinary bladder *per rectum* has seldom been described but in the present study diagnostically significant changes were apparent in almost one third of the animals. In five cases the presence of a bladder mass could be detected, as has been described by Butozan and Mihajlovic (1959), and in three markedly haematuric animals there was massive distention of the bladder, presumably due to obstruction of the urethra by large blood clots. However, it was not possible to appreciate diffuse thickening of the wall of the bladder, as has been reported by Craig (1930).

The haematological changes observed were similar to those recorded by previous authors and were typical of a

haemorrhagic anaemia in that, the extent of depression of erythrocyte counts and haemoglobin concentrations and the degrees of macrocytosis and hypochromasia were related to the severity of haematuria. Thus, although the anaemia was normocytic and normochromic in the majority of cases, which is in agreement with the findings of Rosenberger (1971), there were also a few animals,with severe anaemia,in which there was marked macrocytosis and hypochromasia as recorded by Forero (1960). Leukocyte counts were usually within the normal range and leukopaenia was confined to animals with severe anaemia as has been reported by Rosenberger (1971). The presence of marked neutrophilia in one animal (U1) with chronic suppurative pyelonephritis accords with the statement by Pamukcu (1955) that neutrophilia is occasionally seen in animals which develop secondary bacterial infections of the urinary tract.

In the present study, the only significant change in blood biochemistry was depression of plasma albumin levels which, in individual animals, were directly related to the severity of anaemia. This finding agrees with those of Singh and others (1973) but,in contrast with the results of these authors,the values for plasma calcium and inorganic phosphate were within the normal range,which is consistent with the findings of Georgiev (1957) and Forero (1960).

The pathological findings in this study are similar to those which have been reported elsewhere. Haemangiomas and transitional cell carcinomas were the pre-eminent neoplasms

identified, as in the pathological studies of Pamukcu (1955, 1957),and the major non-neoplastic lesions of cystitis and pyelonephritis are the same as those described by various authors including Bankier (1943) and Nandi (1969).

SECTION III

CLINICAL ASPECTS OF LYMPHOSARCOMA

RESULTS

Clinical examination of animals which were subsequently pathologically confirmed to be affected by lymphosarcoma revealed that the majority of cases could be attributed to three clinical syndromes;

(i) a multicentric form which is characterised by bilaterally symmetrical enlargement of superficial lymph nodes or,less frequently, and only in older animals, by enlargement of localised groups of lymph nodes,

(ii) a thymic form which is characterised by the presence of a cervical and, or anterior thoracic mass with the resultant effects of obstruction of the thoracic inlet by this mass, and

(iii) a skin form which is characterised by nodular infiltration of the skin by neoplastic tissue and bilaterally symmetrical superficial lymph node enlargement.

In addition, a small number of animals exhibited clinical signs which were sufficiently different from the majority of the cases that they could not be attributed to any one of the three syndromes and thus had to be considered as individual entities.

(i) Multicentric Form of Lymphosarcoma

Case Histories

A history was available for all 28 animals. Generalised enlargement of the superficial lymph nodes had been observed in 21 animals, including all those aged less than one year but only three of those aged over one year. Localised lymph node enlargement was observed in the remaining seven animals and was confined to one or both superficial cervical nodes (M23, M24, M25, M27), the right parotid and mandibular nodes (M22), the inguinal nodes (M19) and the mammary nodes (M28). In most cases the lymph node enlargement had only been noticed a few days prior to referral but occasionally, particularly in older animals, gradual enlargement had been observed over several weeks. Loss of condition had occurred in six animals (M19, M23, M24, M26, M27, M28) prior to lymph node enlargement. There were seven males and 21 females ranging in age from two weeks to 11 years (Table 30).

Presenting Signs

Generalised, bilaterally symmetrical enlargement of the superficial lymph nodes was the presenting sign in 23 animals. In the remaining five, all of which were aged one year or older, the presenting sign was regional lymph node enlargement.

Clinical Signs

The demeanour and condition of the animals was extremely variable. Superficial lymph node enlargement was evident on visual examination of all the animals and on

TABLE 30

Multicentric Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Lymph Node Enlargement *							Pallor	Heart/Respiratory Rates Respiratory Signs
			Mandibular	Parotid	Deep Cervical	Superficial Cervical	Subiliac	Superficial Inguinal or Mammary	Medial Iliac		
M1	2w Char x	Moderate Bright	+++	+++	++	+++	+++	+++	-	-	100/50 Hyperpnoea
M2	2w Gall x	Good Bright	+++	+++	+++	+++	+++	+++	-	-	100/70 Hyperpnoea
M3	2w Her x	Poor Dull	+++	++	++	+++	+++	+++	-	+	100/25
M4	2w Ayr	Moderate Bright	+++	+	+	+++	+++	+++	-	-	160/50 Hyperpnoea
M5	3w Sh	Poor Bright	+	-	-	+++	++	++	-	-	80/40 Hyperpnoea
M6	6w AA x	Moderate Bright	+++	++	++	+++	+++	+++	-	+	80/30
M7	3m AA	Poor Bright	++	+++	+	+++	+++	+	-	++	100/120 Dyspnoea Occasional Cough
M8	3m AA x	Good Dull	+++	++	+	++	++	++	-	++	90/90 Hyperpnoea
M9	3m Her	Moderate Bright	+++	+++	+++	+++	+++	+	-	++	90/30
M10	4m Ayr	Poor Dull	+++	+++	-	++	++	++	-	-	100/25

TABLE 30(continued)

Multicentric Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Lymph Node Enlargement *								Heart/Respiratory Rates Respiratory Signs
			Mandibular	Parotid	Deep Cervical	Superficial Cervical	Subiliac	Superficial Inguinal or Mammary	Medial Iliac	Pallor	
M11	4m Sh x	Poor Dull	+++	-	-	+++	+++	+++	-	+++	120/40 Hyperpnoea
M12	4m Her x	Moderate Bright	+++	++	+++	+++	+++	+++	-	-	100/40 Hyperpnoea Occasional cough
M13	5m Gall x	Poor Dull	+++	++	++	+++	+++	++	-	+++	90/120 Hyperpnoea
M14	5m Her x	Poor Dull	+++	++	++	+++	+++	++	-	++	100/25 Hyperpnoea
M15	6m AA x	Good Bright	+++	-	++	+++	+++	+++	-	-	110/80 Hyperpnoea
M16	8m AA x	Poor Bright	+++	+++	+++	+++	++	++	-	+	90/70 Hyperpnoea Occasional cough
M17	12m Her	Poor Dull	+++	+	+++	+++	+++	+++	-	+++	110/70 Hyperpnoea
M18	12m Sh x	Poor Bright	+++	-	-	+++	+++	++	-	-	80/40 Hyperpnoea Occasional cough
M19	12m Gall	Moderate Bright	-	-	-	R++	-	R+++	-	-	70/40
M20	18m Ayr	Moderate Bright	+++	+++	+++	+++	+++	+++	+++	+	110/20

TABLE 30 (continued)

Multicentric Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Lymph Node Enlargement *							Pallor	Heart/Respiratory Rates Respiratory Signs
			Mandibular	Parotid	Deep Cervical	Superficial Cervical	Subiliac	Superficial Inguinal or Mammary	Medial Iliac		
M21	18m Her x	Poor Bright	+++	++	++	+++	+++	+++	+++	+	80/50 Hyperpnoea Occasional cough
M22	21m AA	Poor	R+++	R+++	-	R+++	-	+++	R+++	-	120/15 Snoring Occasional cough Purulent nasal discharge
M23	2y Fries	Moderate Dull	-	++	-	+++	+++	++	+++	-	80/50 Hyperpnoea
M24	2y Fries	Poor Dull	++	-	-	++	++	-	-	++	80/40 Hyperpnoea Occasional cough Rhonchi
M25	4y Fries	Moderate Bright	-	-	+++	R+++	-	-	R++	-	80/40
M26	5y Fries	Poor Dull	+++	-	-	+++	+++	+++	+++	+++	100/15
M27	5y Ayr	Poor Dull	-	-	-	++	L+++	-	+++	+	80/30 Occasional cough
M28	11y Fries	Poor Dull	-	-	-	-	-	+++	+++	-	70/20 Occasional cough

palpation the nodes were firm, smooth, painless, mobile and not hot to the touch (Figure 23). There was also palpable enlargement of lymph nodes which cannot normally be detected; the deep cervical and parotid nodes were enlarged in most cases (Figures 24 and 25) and, occasionally, enlargement of the popliteal and haemal nodes was evident. Enlargement of abdominal lymph nodes, particularly the medial iliac nodes, was evident in eight of the nine older animals on which rectal examination was possible (Table 30).

Respiratory signs including tachypnoea, hyperpnoea, coughing and, on auscultation, harsh respiratory sounds were common but adventitious lung sounds were rare. Pallor of the mucosae and tachycardia were frequently evident but other signs of cardiovascular dysfunction were seldom encountered. Other clinical signs which were occasionally detected included pyrexia (M1, M2, M9, M11, M16, M21, M26), diarrhoea (M3, M21, M24), ruminal tympany (M8) and liver enlargement (M8, M11, M17).

Haematology

The results of haematological examinations performed on each individual animal on admission are recorded in Appendix 3. The mean values of packed cell volume and haemoglobin concentration were outwith the lower extreme of the normal range and the mean erythrocyte count was just within the normal range (Table 31). However there was wide variation in the individual erythrocyte parameters and 32 per cent of the animals were anaemic (packed cell volume < 25%, haemoglobin concentration < 8g/100mls). The mean values of mean cell volume and mean cell haemoglobin

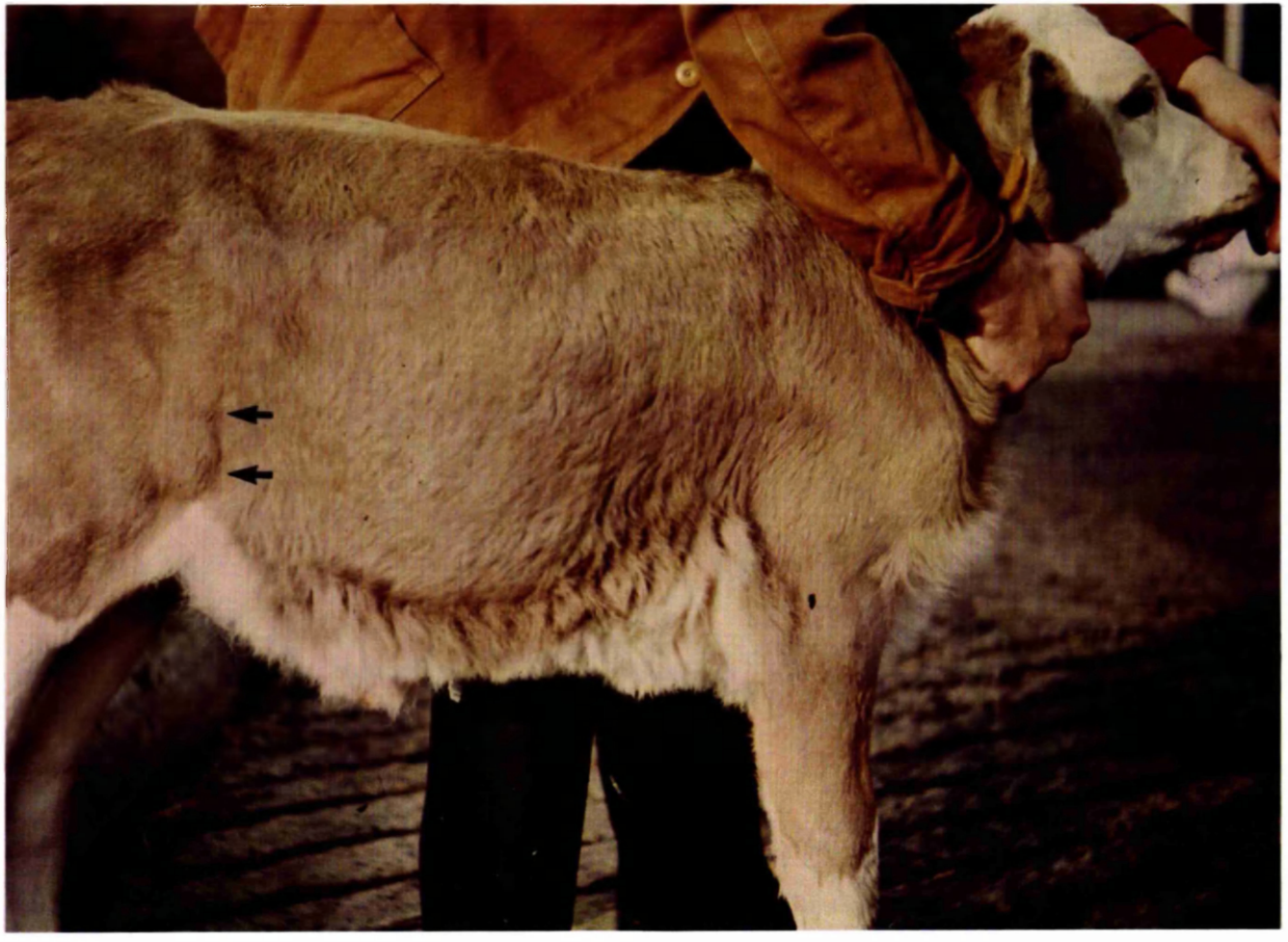


FIGURE 23 Multicentric lymphosarcoma : Visible enlargement of the right subiliac lymph node

FIGURE 24 Multicentric lymphosarcoma : Visible enlargement of the right parotid lymph node

FIGURE 25 Multicentric lymphosarcoma : Massive enlargement of the right mandibular and parotid lymph nodes

TABLE 31

Multicentric Lymphosarcoma

Summary of Haematological Parameters

on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	28	26.1 ± 6.3	27 - 35
Haemoglobin (g/100mls)	25	8.1 ± 1.9	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	25	5.66 ± 1.47	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	25	31.5 ± 3.1	30 - 36
Mean Cell Volume (μ^3)	25	47.0 ± 9.1	40 - 60
Leukocyte Count ($x10^3/mm^3$)	28	26.6 ± 35.3	7 - 10

concentration were within the normal range (Table 31) and macrocytosis or hypochromia were infrequently encountered in individual animals.

The mean total leukocyte count was markedly raised (Table 31) although there was extreme variation in individual total leukocyte counts as is indicated by the very large standard deviation of the mean value. 16 animals (57%) had leukocytosis (>10.0 x 10^3 leukocytes/mm^3) of which eight were markedly leukocytotic (>20.0 x 10^3 leukocytes/mm^3). In 12 of the animals which had leukocytosis, this was due to lymphocytosis or, in one case (M17), monocytosis and in these animals the absolute lymphocyte or monocyte count ranged between 11.2 x 10^3 and 106.9 x 10^3 per mm^3 (Figure 26). In addition, lymphoblasts or monoblasts were present in the blood of 14 animals, including all those in which there was marked leukocytosis.

Biochemistry

The results of blood biochemical analysis performed on each individual animal on admission are recorded in Appendix 3. The mean values of the majority of the plasma constituents measured fell within the normal range (Table 32). The only exceptions were blood urea and bilirubin both of which were marginally elevated. Blood urea was raised in 11 animals (39%) and bilirubin in 14 (50%) but in neither case were any individual levels markedly elevated. Plasma albumin levels were depressed (<25 g/l) in 10 animals but were only markedly depressed (<20 g/l) in half of these cases.

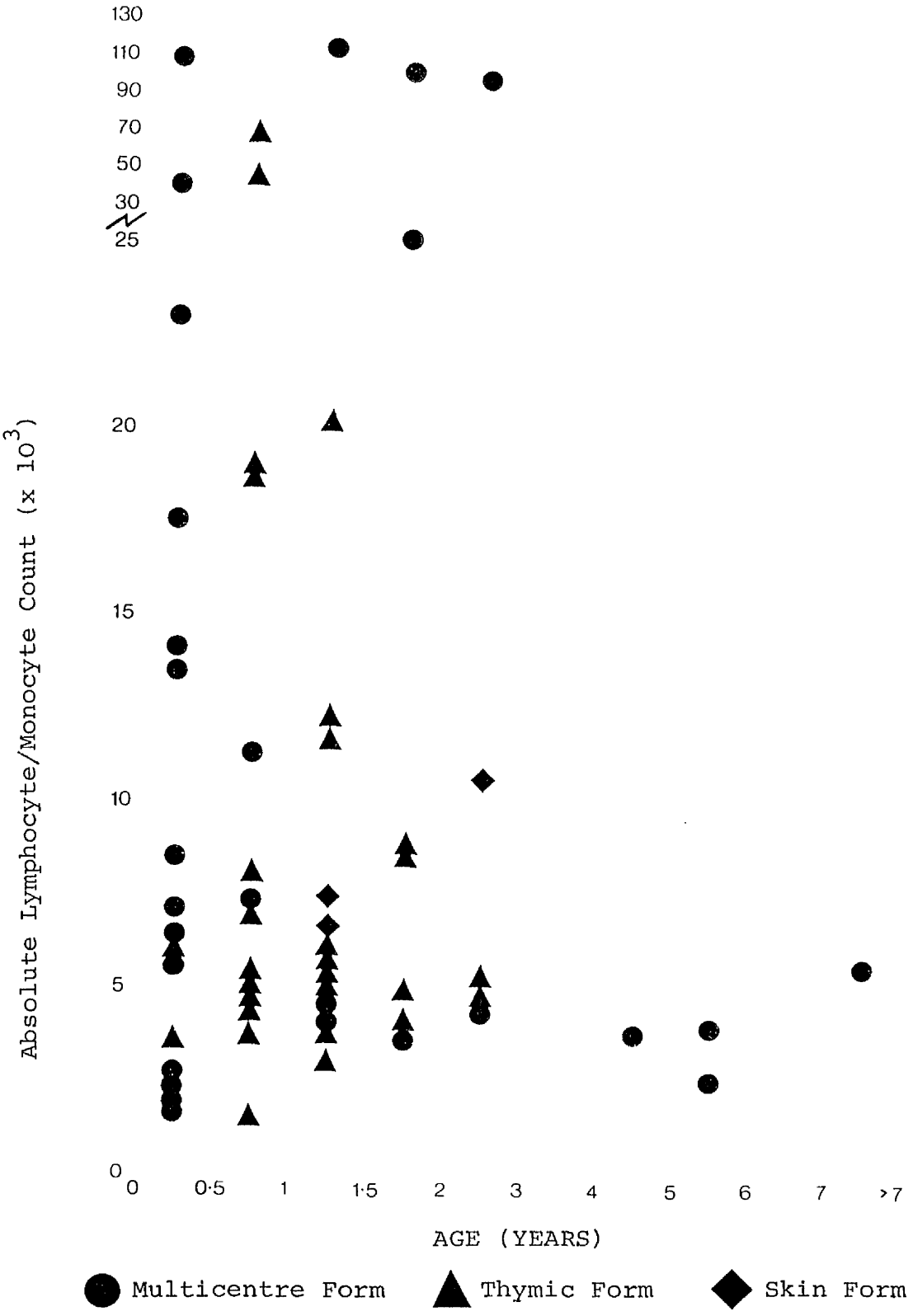


FIGURE 26

Absolute lymphocyte/monocyte Count of Animals Affected by Multicentric, Thymic and Skin Forms of Lymphosarcoma.

TABLE 32

Multicentric Lymphosarcoma

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	28	8.5 ± 5.1	0 - 8.3
SODIUM mmol/l	28	142 ± 6	136 - 151
POTASSIUM mmol/l	28	4.8 ± 1.1	3.2 - 5.8
CHLORIDE mmol/l	28	103 ± 8	96 - 111
CALCIUM mmol/l	27	2.35 ± 0.40	2.29 - 3.08
MAGNESIUM mmol/l	27	0.75 ± 0.23	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	28	2.74 ± 0.94	1.13 - 2.84
BILIRUBIN µmol/l	28	9 ± 7	0 - 8
ALKALINE PHOSPHATASE IU/l	28	85 ± 62	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	27	136 ± 167	0 - 200
ALANINE AMINOTRANSFERASE IU/l	28	26 ± 19	0 - 40
TOTAL PROTEIN g/l	28	70 ± 11	50 - 90
ALBUMIN g/l	28	25 ± 6	25 - 40
GLOBULIN g/l	28	45 ± 12	25 - 55

Pathological Findings

All the superficial lymph nodes were totally replaced by lymphosarcoma in the majority of animals. In the remaining cases (M19, M22, M24, M25, M27, M28) localised groups of superficial lymph nodes were replaced by tumour tissue and the other superficial nodes, although not markedly enlarged, were frequently found to be infiltrated to a lesser extent. In all but two of the animals (M19, M24) the lymph nodes of the thoracic and abdominal cavities were also heavily infiltrated by lymphosarcoma.

Diffuse infiltration and, or nodular masses of lymphosarcoma were present in the liver, spleen and kidneys of the vast majority of the animals. The lung parenchyma was infiltrated in five animals (M6, M17, M21, M26, M27). In addition tumour tissue was found in a variety of other sites including the duodenum (M11, M23, M27) the abomasum and omasum (M22, M27) the myocardium and pericardium (M22) and the cornea (M17).

(ii) Thymic Form of Lymphosarcoma

Case Histories

A history was available for all 30 animals. A mass or oedematous swelling had developed in the ventral neck and, or in the presternal area in 25 animals over a period ranging between two days and several weeks prior to admission. Loss of condition had also been observed in many of these animals. In the five remaining animals the farmer had noticed enlargement of the superficial cervical lymph nodes (T6, T12) rumenal tympany (T23, T30) or severe respiratory signs (T16.).

There were five males and 25 females ranging in age from four months to two years (Table 33).

Presenting Signs

The presenting feature in 26 animals was an obvious swelling in the presternal area and, or the ventral aspect of the neck (Figure 27). The remaining four animals presented with massive enlargement of the superficial cervical lymph nodes (T6, T12), respiratory distress (T16) or massive rumenal tympany (T23).

Clinical Signs

In 22 of the 26 animals in which there was an obvious swelling in the presternal area and, or ventral neck, the swelling was composed of a firm mass which in 12 was surrounded by oedema. Oedema was so extensive in the remaining four animals (T5, T21, T27, T29) that detailed examination of the area was precluded. Less frequently oedema was also present in other dependant areas (Table 33). Enlargement of the superficial cervical lymph nodes was visibly or palpably evident in 26 animals, including those in which this was the presenting sign, but enlargement of other

FIGURE 27 Thymic lymphosarcoma : Swelling of the ventral aspect of the neck and presternal area

TABLE 33

Thymic Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Presternal Ventral Neck		Oedema* Other Sites	Distension of Jugular Veins	Lymph Node** Enlargement		Cardiac Muffling	Pallor	Ruminal Tympany	Heart/Respiratory Rates Respiratory Signs
			Mass	Oedema			Superficial Cervical	Other				
T1	4m Gall	Moderate Dull	- +++	- -	-	+++	R+	-	-	-	-	120/40 Hyperpnoea
T2	4m Her	Moderate Bright	++ +++	- -	-	+++	+	-	-	-	+	100/40 Dyspnoea Frequent cough
T3	6m Fries	Poor Dull	++ -	- -	-	++	++	-	-	-	-	55/25
T4	6m Fries	Good Dull	- ++	+++ +	SM++	+	++	M L++ R++	+	-	++	100/20 Hyperpnoea Occasional cough
T5	7m A.A.	Poor Dull	- -	+++ -	SM++	+++	+++	M+++ SI+++ IN+++	-	+	++	90/40 Hyperpnoea
T6	8m Jer	Poor Bright	-	-	-	+++	+++	-	++	-	++	100/20
T7	8m Ayr	Poor Dull	++ -	+++ ++	-	+++	+++	M+	L++	-	-	100/60 Dyspnoea Occasional cough Expiratory grunt
T8	8m Sh x	Good Bright	++ +++	- -	-	-	L+	SI L+	-	-	++	80/75 Hyperpnoea
T9	8m A.A.	Good Dull	++ +++	- -	SM++	+++	+	M+ MAM+++	-	+	+	80/40 Hyperpnoea Occasional cough
T10	8m Her x	Good Bright	+++ -	+++ -	-	+++	+++	H+	-	+++	-	120/20

TABLE 33 (continued)

Thymic Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Presternal Ventral Neck		Oedema* Other Sites	Distension of Jugular Veins	Lymph Node** Enlargement		Cardiac Muffling	Pallor	Ruminal Tympany	Heart/Respiratory Rates Respiratory Signs
			Mass	Oedema			Superficial Cervical	Other				
T11	8m Fries	Moderate Bright	+++ -	+++ -	-	+++	+++	IN+	L+	-	-	85/25 Hyperpnoea
T12	10m Ayr	Poor Bright	+ -	- -	-	+	+++	M+++ PR++ SI R++	L+	-	-	100/30
T13	10m Ayr	Poor Dull	- ++	- -	-	+++	++	-	-	-	+	70/40 Dyspnoea Occasional cough Expiratory grunt
T14	10m Her x	Moderate Dull	++ -	+ +++	SM+++	++	++	-	+++	-	+++	90/60 Dyspnoea
T15	12m Her x	Good Dull	++ +++	++ -	SM+++	+++	+++	-	+	-	++	90/50
T16	12m Ayr	Moderate Dull	- -	- -	-	+	+	-	L+++ R+++	-	-	100/50 Dyspnoea Expiratory grunt Respiratory sounds absent left lung
T17	12m A.A.	Good Dull	+++ +++	+ -	-	-	-	-	-	-	++	9/25
T18	12m A.A.x	Good Bright	+++ ++	+++ ++	SM+++	+++	+++	M+++	-	-	++	80/25
T19	12m Char	Moderate Bright	- +++	- -	-	+++	-	-	-	-	+	90/30 Occasional cough
T20	14m Her x	Good Bright	+++ +	- -	-	+++	L+ R+++	H+	-	-	++	80/50 Hyperpnoea

TABLE 33 (continued)

Thymic Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Presternal Ventral Neck		Oedema* Other Sites	Distension of Jugular Veins	Lymph Node** Enlargement		Cardiac Muffling	Pallor	Ruminal Tympany	Heart/Respiratory Rates Respiratory Signs
			Mass	Oedema			Superficial Cervical	Other				
T21	15m Her x	Moderate Dull	- -	+++ ++	SM+++ AW+++	+++	-	SI R++	R++	-	+++	100/30 Dyspnoea Expiratory grunt
T22	15m Ayr	Good Dull	+++ +++	- -	SM++	+++	+	M R++ P R++ H +	L+++	-	+++	110/40 Hyperpnoea
T23	15m Ayr	Moderate Bright	+ -	- -	-	+++	-	MAM++	L++	-	+++	95/30 Hyperpnoea
T24	18m Her x	Poor Dull	+++ -	- -	-	+++	L++ R+++	-	R+	+	-	70/50 Hyperpnoea Frequent cough Expiratory grunt
T25	18m Fries	Good Bright	+++ +++	+ +	-	-	R++	-	-	-	-	80/40 Hyperpnoea Frequent cough
T26	18m Fries	Moderate Dull	++ +++	- -	-	+++	+	-	-	-	++	70/30 Hyperpnoea Occasional cough
T27	21m Fries	Poor Dull	- -	+++ -	AW+++ FL+++	+++	+	-	+++	+	-	150/50 Hyperpnoea Expiratory grunt
T28	21m Fries	Good Bright	++ -	+++ -	SM+++ AW+++	+++	L++ R+++	-	R+++	-	++	80/40 Occasional cough
T29	2y Ayr	Good Bright	- -	+++ -	SM+++ AW++	+++	L+++ R++	-	L+++ R+	-	+++	90/25
T30	2y Ayr	Moderate Bright	+ ++	++ -	-	+++	+++	U+++	R++	-	++	70/20

* SM = submandibular, FL = forelimbs, AW = ventral abdominal wall
** M = mandibular, MAM = mammary, H = haemal, P = parotid, SI = subiliac, IN = inguinal
U = uterine, R = right only, L = left only.

superficial nodes was less frequent, being present in 14 cases (Table 33).

Distention of the jugular veins was almost always present but pulsation was rarely apparent. On auscultation, bilateral or unilateral muffling of cardiac sounds was evident in 16 animals, and, on percussion, decreased thoracic resonance was detected in all but three of these (T10, T11, T19). Tachycardia and mucosal pallor were occasionally present but seldom marked.

Dyspnoea and, or frequent coughing were present in seven animals and many of the others were tachypnoeic and hyperpnoeic. Although rumenal tympany was present in 18 cases it was severe in only five (T14, T21, T22, T23, T29). The consistency of the faeces was variable and seven animals (T8, T19, T23, T24, T27, T28, T30) were diarrhoeic. However, despite the normal consistency of the faeces in most of the remaining animals they were frequently scanty in amount. Other clinical signs which were less frequently observed included pyrexia (T7, T16, T22), ataxia (T1, T20) and straining due to massively enlarged uterine lymph nodes (T30).

Haematology

The results of haematological examinations performed on each individual animal on admission are recorded in Appendix 3. The mean values of packed cell volume, haemoglobin concentration, erythrocyte count, mean cell volume and mean cell haemoglobin concentration were within

the normal range (Table 34). Only two animals (T5, T10) were anaemic (packed cell volume <25%, haemoglobin concentration <8g/100mls).

The mean total leukocyte count was raised (Table 34) although there was considerable variation in individual total leukocyte counts as is indicated by the large standard deviation of the mean value. 14 animals (47%) had leukocytosis (>10.0 x 10^3 leukocytes/mm^3) of which five were markedly leukocytotic (>20.0 x 10^3 leukocytes/mm^3). In seven of the animals which had leukocytosis, this was due to lymphocytosis and, in these animals, the absolute lymphocyte count ranged between 11.6 and 66.7 x 10^3 per mm^3 (Figure 26). In addition, lymphoblasts were present in the blood of 14 animals, including all those in which there was marked leukocytosis.

Biochemistry

The results of blood biochemical analysis performed on each individual animal on admission are recorded in Appendix 3. The mean values of the majority of the plasma constituents fell within the normal range (Table 35). The only exceptions were bilirubin and aspartate amino transferase both of which were marginally elevated. Bilirubin was raised in 19 animals but none of the individual levels were markedly elevated. Aspartate aminotransferase was raised in 14 animals and markedly elevated in two cases (T21, T23). Plasma albumin levels were depressed (<25g/l) in 12 animals but were only markedly depressed (<20g/l) in a quarter of these cases.

TABLE 34

Thymic Lymphosarcoma
Summary of Haematological Parameters
on Admission

PARAMETER	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
Packed Cell Volume (%)	30	31.1 ± 5.6	27 - 35
Haemoglobin (g/100mls)	24	9.6 ± 2.2	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	24	6.74 ± 1.57	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	24	32.2 ± 3.2	30 - 36
Mean Cell Volume (μ^3)	24	46.3 ± 6.8	40 - 60
Leukocyte Count ($x10^3/mm^3$)	30	14.2 ± 13.1	7 - 10

TABLE 35

Thymic Lymphosarcoma

Summary of Biochemical Parameters on Admission

PLASMA CONSTITUENT	NUMBER OF ANIMALS	MEAN ± STANDARD DEVIATION	NORMAL RANGE
UREA mmol/l	29	7.1 ± 5.5	0 - 8.3
SODIUM mmol/l	30	142 ± 7	136 - 151
POTASSIUM mmol/l	30	4.3 ± 0.6	3.2 - 5.8
CHLORIDE mmol/l	30	98 ± 7	96 - 111
CALCIUM mmol/l	28	2.37 ± 0.20	2.29 - 3.08
MAGNESIUM mmol/l	28	0.73 ± 0.31	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	28	2.46 ± 0.66	1.13 - 2.84
BILIRUBIN µmol/l	29	11 ± 7	0 - 8
ALKALINE PHOSPHATASE IU/l	30	61 ± 25	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	30	159 ± 113	0 - 200
ALANINE AMINOTRANSFERASE IU/l	30	34 ± 20	0 - 40
TOTAL PROTEIN g/l	30	80 ± 11	50 - 90
ALBUMIN g/l	30	26 ± 5	25 - 40
GLOBULIN g/l	30	54 ± 11	25 - 55

Pathological Findings

The thoracic and, or cervical portions of the thymus were partially or wholly replaced by lymphosarcoma in every animal and with one exception (T17) there was also evidence of lymph node involvement. The superficial cervical lymph nodes were infiltrated or totally replaced by tumour tissue in 26 animals (87%) and other superficial lymph nodes were similarly affected in 23 animals (77%). The lymph nodes of the thoracic cavity, particularly the mediastinal nodes, were infiltrated in the vast majority of cases (90%) but involvement of abdominal lymph nodes was less frequently found (57% of cases).

Within the thoracic cavity local extension of the tumour had occurred in a number of animals to involve the lung parenchyma (T1, T7, T16, T23, T24, T27) or myocardium (T22). Diffuse and, or nodular infiltration was evident in the liver of 11 animals (37%), the spleen of 7 animals (23%) and the kidneys of three animals (10%). In addition, tumour tissue was found in a variety of other sites including the vertebrae and spinal canal (T1, T20, T23), the duodenum (T7) and the abomasum and omasum (T8).

(iii) Skin Form of Lymphosarcoma

Case Histories

A history was available for all three animals and in each case the farmer had observed the development of nodular swellings in the skin over a period of several weeks. The animals were all castrated males which were aged between 15 and 24 months.

Presenting Signs

In each case the presenting sign was the presence of numerous greyish-white raised nodules and plaques which were widely scattered over the body surface.

Clinical Signs

The skin lesions were circular and well demarcated except where they were extremely numerous when they tended to coalesce. In two animals (S1, S2) the lesions were exuberant producing fungating plaques above the skin surface, but in the remaining animal (S3) they were mainly subcutaneous, smooth, firm and nodular (Figure 28). The surfaces of the lesions were fragile and haemorrhage was readily induced, even by mild trauma.

In addition, generalised, bilaterally symmetrical, superficial lymph node enlargement was present in all three animals and rectal examination revealed enlargement of the medial iliac lymph nodes in two cases (S1, S3). On admission, subcutaneous oedema was evident in two animals (S2, S3) and subsequently this feature also developed in the third case. The major clinical findings in each case are summarised in Table 36.

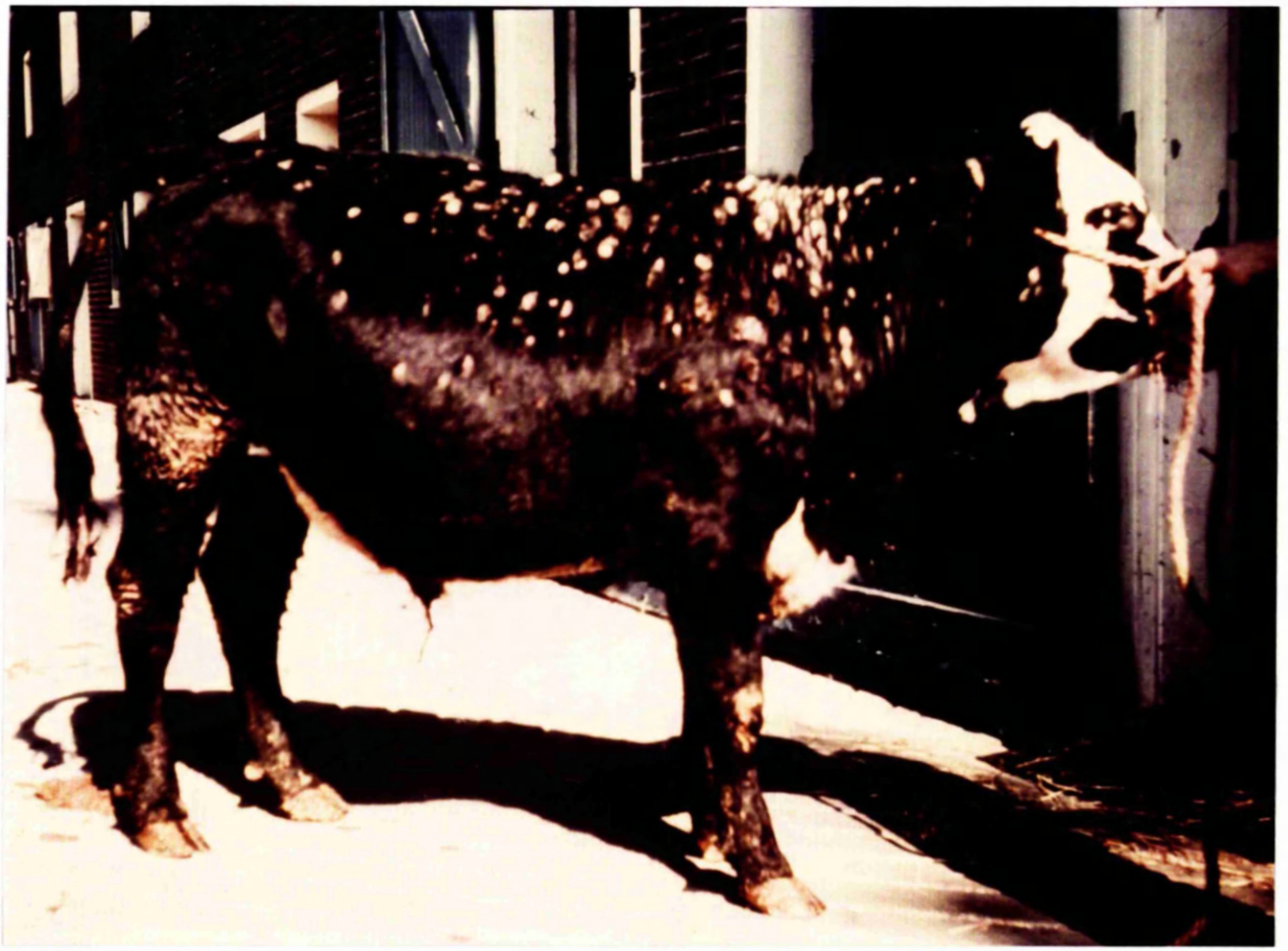


FIGURE 28 Skin lymphosarcoma : Nodular skin lesions.
Note the visibly enlarged subiliac lymph node

TABLE 36

Skin Lymphosarcoma - Summary of Major Clinical Findings

Case No.	Age and Breed	Condition and Demeanour	Distribution of Skin Lesions	Lymph Node Enlargement						Pallor (PCV%)	Heart/Respiratory Rates Respiratory Signs
				Oedema	Mandibular	Superficial Cervical	Subiliac	Superficial Inguinal or Mammary	Medial Iliac		
S1	15m Her x	Moderate Bright	Whole body particularly flanks, ventral abdominal wall, perineum, head and neck.	-	+++	+++	+++	+++	+++	-	70/35 Hyperpnoea
S2	15m Her x	Poor Bright	Whole body particularly dorsally and flanks.	Prester-nal +++ Ventral Abdominal Wall ++ Limbs ++	+++	+++	+++	+++	-	-	120/70 Hyperpnoea
S3	2y Fries	Poor Dull	Whole body particularly ventral abdominal wall, groins and perineum.	Subman-dibular ++ Prester-nal ++ Ventral Abdominal Wall +++	+++	+++	+++	+++	+++	+++	100/90 Hyperpnoea

Haematology

The results of haematological examinations performed on each of the three animals on admission are recorded in Table 37. Two of the animals (S2, S3) were anaemic (packed cell volume <25%, haemoglobin concentration <8g/100mls) and in one of these (S2) there was a slight degree of hypochromia. These animals also had leukocytosis which in one (S2) was due to neutrophilia and in the other (S3) lymphocytosis (Figure 26). In addition, lymphoblasts were present in the blood of this latter case.

Biochemistry

The results of blood biochemical analysis performed on each of the three animals on admission are recorded in Table 38. All three animals had depressed levels of plasma albumin. In addition, one animal (S3) had elevated levels of bilirubin, aspartate and alanine amino transferase, inorganic phosphate and blood urea.

Pathological Findings

Multicentric lymphosarcoma with extensive involvement of the skin was present in all three animals. Subcutaneous infiltration by the tumour had produced numerous nodules ranging in size between two and 10 centimetres in diameter. The nodules were mainly subcutaneous in one animal (S3) but in the other two there was infiltration through the skin and ulceration of the skin surface. The superficial lymph nodes were replaced by tumour tissue in all three animals and most of the thoracic and abdominal lymph nodes were involved.

TABLE 37

Skin Lymphosarcoma

Haematological Parameters on Admission

PARAMETER	CASE NUMBER S1	CASE NUMBER S2	CASE NUMBER S3	NORMAL RANGE
Packed Cell Volume (%)	29.5	24	19	27 - 35
Haemoglobin (g/100mls)	9.9	6.7	6.0	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	7.60	5.71	3.55	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	33.6	27.9	31.5	30 - 36
Mean Cell Volume (μ^3)	39	42	54	40 - 60
Leukocyte Count ($x10^3/mm^3$)	9.9	12.5	14.3	7 - 10

TABLE 38

Skin Lymphosarcoma

Biochemical Parameters on Admission

PLASMA CONSTITUENT	CASE NUMBER S1	CASE NUMBER S2	CASE NUMBER S3	NORMAL RANGE
UREA mmol/l	2.7	1.9	9.2	0 - 8.3
SODIUM mmol/l	138	133	140	136 - 151
POTASSIUM mmol/l	3.3	3.6	4.7	3.2 - 5.8
CHLORIDE mmol/l	104	96	90	96 - 111
CALCIUM mmol/l	2.45	2.56	2.39	2.29 - 3.08
MAGNESIUM mmol/l	0.74	0.61	0.89	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	2.39	1.30	3.22	1.13 - 2.84
BILIRUBIN µmol/l	5	7	30	0 - 8
ALKALINE PHOSPHATASE IU/l	36	12	43	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	59	191	856	0 - 200
ALANINE AMINOTRANSFERASE IU/l	33	38	61	0 - 40
TOTAL PROTEIN g/l	74	77	73	50 - 90
ALBUMIN g/l	23	19	22	25 - 40
GLOBULIN g/l	51	58	51	25 - 55

Diffuse or nodular infiltration of the liver (S1, S3) the spleen (S1, S3) and the kidneys (S1, S2, S3) was found and in case S3 there was diffuse infiltration of the myocardium of the atria and ventricles. Ulceration of the abomasal mucosa was also evident in this latter animal and the raised edges of the ulcers were found to be composed of tumour tissue.

(iv) Atypical Clinical Forms of Lymphosarcoma

In three animals there was no clinical evidence of lymph node enlargement or a thymic mass and thus each case is described individually.

Case Number AL1

Case History

This was a four year old Friesian cow which had gradually lost condition over a period of several months.

Clinical Signs

The animal was very thin, dull and afebrile. Marked pallor of the mucosae was evident but there was no tachycardia, the heart rate being 70 beats per minute. Apart from an occasional, harsh non-productive cough, there were no respiratory abnormalities. There was anorexia and ruminal stasis and only scant quantities of faeces were passed. None of the superficial lymph nodes were enlarged and no abnormalities were detected on rectal examination.

Haematology

Haematological examination confirmed the presence of anaemia which was normocytic and normochromic (Table 39). The total leukocyte count was raised but the differential count (78.5% neutrophils, 21.5% lymphocytes) indicated that the leukocytosis was the result of neutrophilia.

Biochemistry

The only significant biochemical abnormalities were the presence of severe hypoalbuminaemia (Table 40) and a mild proteinuria (54 mg/100mls).

TABLE 39

Atypical Clinical Forms of Lymphosarcoma

Haematological Parameters on Admission

PARAMETER	CASE NUMBER AL1	CASE NUMBER AL2	CASE NUMBER AL3	NORMAL RANGE
Packed Cell Volume (%)	20	29	25	27 - 35
Haemoglobin (g/100mls)	6.5	-	6.3	9 - 12
Erythrocyte Count ($x10^6/mm^3$)	3.49	-	3.73	5 - 8
Mean Cell Haemoglobin Concentration (g/dl)	32.5	-	25.2	30 - 36
Mean Cell Volume (μ^3)	57	-	67	40 - 60
Leukocyte Count ($x10^3/mm^3$)	13.9	16.2	17.4	7 - 10

TABLE 40

Atypical Clinical Forms of Lymphosarcoma

Biochemical Parameters on Admission

PLASMA CONSTITUENT	CASE NUMBER AL1	CASE NUMBER AL2	CASE NUMBER AL3	NORMAL RANGE
UREA mmol/l	6.6	7.6	16.8	0 - 8.3
SODIUM mmol/l	130	128	139	136 - 151
POTASSIUM mmol/l	3.6	3.1	5.3	3.2 - 5.8
CHLORIDE mmol/l	95	97	77	96 - 111
CALCIUM mmol/l	2.05	1.70	2.38	2.29 - 3.08
MAGNESIUM mmol/l	0.49	0.21	1.23	0.65 - 1.39
INORGANIC PHOSPHATE mmol/l	2.42	1.81	2.17	1.13 - 2.84
BILIRUBIN µmol/l	7	12	85	0 - 8
ALKALINE PHOSPHATASE IU/l	85	43	341	4 - 127
ASPARTATE AMINOTRANSFERASE IU/l	164	23	123	0 - 200
ALANINE AMINOTRANSFERASE IU/l	16	18	11	0 - 40
TOTAL PROTEIN g/l	56	104	79	50 - 90
ALBUMIN g/l	7	29	16	25 - 40
GLOBULIN g/l	49	95	63	25 - 55

Pathological Findings

There was extensive infiltration of the wall and folds of the abomasum and the wall and leaves of the omasum by lymphosarcoma. The lymph nodes associated with the abomasum were completely replaced by tumour tissue and measured up to 12 centimetres in diameter. A single polyp measuring two centimetres in diameter was found in the duodenum, approximately two metres from the pylorus,and there was infiltration of the mesenteric lymph nodes. The hepatic and posterior mesenteric lymph nodes were also replaced by tumour tissue. Several small nodules of lymphosarcoma were apparent in the kidneys.

Case Number AL2

Case History

This was a six year old Friesian cow in which anorexia and mild rumenal tympany had developed.

Clinical Signs

The animal was thin, dull, anorexic and slightly pyrexic. Tachypnoea, (respiratory rate, 40 per minute), hyperpnoea and an expiratory grunt were evident and the cow coughed occasionally. On auscultation, harsh respiratory sounds were apparent and rhonchi were heard in the left anteroventral lung field, percussion of which elicited a painful response. Mild rumenal tympany was a constant feature but there were no other clinical abnormalities of the alimentary system. Rectal examination revealed

enlargement of the left kidney and post admission transient haematuria was observed.

Haematology

The packed cell volume was within the normal range (Table 39). The total leukocyte count was raised but the differential count (78% neutrophils, 22% lymphocytes) indicated that the leukocytosis was the result of neutrophilia.

Biochemistry

There were no major blood biochemical abnormalities. However urine examination indicated that there was a moderately severe proteinuria (224 mg/100mls).

Pathological Findings

There was diffuse infiltration of the mediastinum by lymphosarcoma. The left diaphragmatic lung lobe was diffusely infiltrated and there was tumour tissue on the pleural surface of the lungs and on the dorsal thoracic wall. Many of the visceral lymph nodes of the abdomen and thorax were replaced by tumour and the kidneys had numerous tumour nodules projecting from their surfaces.

Case Number AL3

Case History

This was a 12 year old Hereford cross cow which had been profusely diarrhoeic for one week prior to admission.

Clinical Signs

The animal was thin, dull, anorexic and afebrile. The mucosae were jaundiced but there was no palpable enlargement of the liver. A loud bilateral systolic cardiac murmur was evident but tachycardia was not apparent. There was rumenal stasis, the abdomen was reduced in size and a profuse blood tinged diarrhoea was present. None of the superficial lymph nodes were enlarged and no abnormalities could be detected on rectal examination.

Haematology

A macrocytic hypochromic anaemia was present (Table 39). In addition, the total leukocyte count was raised but the differential count (65.5% neutrophils, 34.5% lymphocytes) indicated that the leukocytosis was the result of neutrophilia.

Biochemistry

The significant biochemical abnormalities were severe hypoalbuminaemia and elevated levels of blood urea, bilirubin and alkaline phosphatase (Table 40).

Pathological Findings

Two large nodular masses of lymphosarcoma, 20 centimetres in diameter, were present at the hilus of the liver, replacing the hepatic lymph nodes and involving the duodenum and pancreas. The liver was diffusely infiltrated by lymphosarcoma but also contained discrete tumour nodules up to six centimetres in diameter. The wall of the gall

bladder was diffusely infiltrated. In addition the mediastinal lymph nodes were replaced by tumour tissue.

DISCUSSION

Generalised bilaterally symmetrical enlargement of superficial lymph nodes was the outstanding feature of multicentric lymphosarcoma in animals aged less than one year. This feature and the other clinical signs observed in the present study are similar to those described in the only other detailed descriptions of this form of lymphosarcoma (Theilen and Dungworth, 1965; Theilen and Madewell, 1979). The major haematological abnormalities recognised in this study were also similar to those identified by Theilen and Dungworth (196). Leukaemia was evident in 10 (36%) of the animals in the present study compared with 3 (43%) of the cases examined by Theilen and Dungworth (1965). In addition, anaemia was evident in approximately half the animals in each study. Similarly the biochemical findings in the two studies were comparable, with the exception that there was no evidence of depressed levels of plasma globulins in the present study whereas Theilen and Dungworth (1965) found low plasma globulins in five of the six animals in which this parameter was examined.

In animals aged one year and older, 50 per cent of the animals affected by multicentric lymphosarcoma had regional rather than generalised lymph node enlargement

which is in agreement with Bendixen (1961a) and Marshak and others (1962). However two notable clinical signs, posterior paresis and exopthalmos, were present in a significant proportion of the animals examined by Marshak and others (1962) and Theilen and Madewell (1979), but were not observed in the present study. In addition, involvement of the heart, alimentary tract and epidural space was rarely evident at post mortem examination in the present study, but was frequently found by Marshak and others (1962) and Theilen and Madewell (1979). An explanation of these differences may be related to the high probability that the latter authors were examining cases of enzootic bovine leukosis whereas the animals in the present study were almost certainly examples of sporadic lymphosarcoma in adults (vide infra).

The combination of clinical signs which were found in animals affected by thymic lymphosarcoma were, in most cases, so distinctive that diagnosis rarely presented any difficulty. The clinical and laboratory findings described in this study are generally comparable to those of Dungworth and others (1964) and Theilen and Madewell (1979) who have provided the only other detailed clinical descriptions of this form of lymphosarcoma.

However several aspects of these three studies are worthy of note. In the present study, 28 of the 30 animals had clinically detectable enlargement of one or more superficial lymph nodes and, in 26 cases, enlargement of one or both

superficial cervical lymph nodes was evident. Theilen and Madewell (1979) also identified enlargement of lymph nodes in a high proportion (72%) of cases, but Dungworth and others (1964) state that, in their study, lymph node enlargement was not a prominent feature. In addition, in the present study, enlargement of lymph nodes was considered to be a useful diagnostic feature, particularly in cases in which the thymic mass was situated in the thoracic cavity, whereas Dungworth and others (1964), who found diagnosis difficult when the thymic mass was intrathoracic, considered that lymph node palpation was of no help in reaching a diagnosis In contrast, the results of both these studies indicate that haematological examinations are seldom an aid to diagnosis. Five (17%) of the animals (T5, T7, T9, T13, T20) in the present study showed evidence of leukaemia which is a similar proportion to that found by Dungworth and others (1964) who identified only one leukaemic animal in the eight cases on on which they performed haematological examinations.

Only three cases of the skin form of lymphosarcoma were examined in this study but there was a uniform clinical picture which corresponds in most respects with previous clinical descriptions of this form of the disease. However regression of the skin lesions as described by Bendixen (1961b) and Clegg and Moss (1965) was not observed and the development of subcutaneous oedema, which was a notable clinical sign in the present study, is not a feature recorded by previous authors.

The three clinically atypical cases of lymphosarcoma all of which were adult animals, were essentially examples of multicentric lymphosarcoma affecting viscerial lymph nodes and organs but without involvement of superficial lymph nodes. Marshak and others (1962), in their clinical study of lymphosarcoma in adult cattle, also recorded that in a small proportion of cases (8%) superficial lymphodenopathy was not a feature. Occasionally, other authors have reported examples of clinically atypical cases of lymphosarcoma, including Smith and Anderson (1977) who described nervous signs in a heifer with marked lymphocytosis, in which the only site where a tumour mass could be identified was the brain. These atypical cases obviously present considerable difficulties in clinical diagnosis and unless there are abnormalities such as enlargement of abdominal lymph nodes which can be detected per rectum or haematological evidence of leukaemia, the diagnosis must rely on pathological examination.

Although lymphosarcoma is considered to be amongst the most common of bovine malignancies, there are very few detailed descriptions of the clinical syndromes associated with this neoplasm. The present study provides a comprehensive description of the spectrum of clinical signs which can be observed and indicates that, with rare exceptions, a diagnosis can be achieved solely on the basis of a detailed clinical examination.

CHAPTER 3

AN EPIDEMIOLOGICAL STUDY OF BOVINE NEOPLASIA WITH PARTICULAR REFERENCE TO ALIMENTARY AND URINARY BLADDER NEOPLASMS AND THEIR RELATIONSHIP WITH BRACKEN FERN (PTERIDIUM AQUILINUM)

REVIEW OF THE LITERATURE

Upper Alimentary Squamous Cell Carcinoma

Surveys of bovine neoplasia which have been performed in abbatoirs and at veterinary schools throughout the world indicate that, in general, upper alimentary squamous cell carcinoma of cattle is a rare malignancy. Only nine examples were identified amongst a total of almost 5000 bovine malignancies examined in surveys performed in the United States of America (Davis, Leeper and Shelton, 1933; Monlux and others, 1956; Brandly and Migaki, 1963), Canada (Plummer, 1956), the Netherlands (Misdorp, 1967), the United Kingdom (Cotchin, 1960; Anderson and others, 1969) and India (Nair and Sastry, 1953). However, in two surveys performed in Scotland and South Africa the prevalence of upper alimentary squamous cell carcinoma was much higher with 25 cases being found in the 305 malignancies examined by Trotter (1911) and six cases in the 108 malignancies examined by Jackson (1936). In addition, the German authors, Nieberle and Cohrs (1949) and Kitt (1950), considered that upper alimentary squamous cell carcinoma was not a rare neoplasm and the latter author suggested that it accounted for approximately five per cent of malignancies in cattle. Thus, although upper alimentary squamous cell carcinoma is generally rare it would appear that in certain regions it is relatively common, and during the last 30 years two such areas have been described in detail.

Plowright (1955) and Plowright and others (1971) described a 'high incidence' of upper alimentary squamous cell carcinoma amongst Zebu cattle indigenous to the Nasampolai valley in the Narok district of Kenya Masailand. In affected animals, which were aged four to 15 years, the carcinoma appeared to be multicentric in origin with the involvement of two main sites, the anterior dorsal sac of the rumen and the oesophagus. Two other neoplasms were also identified in a proportion of the cases; upper alimentary papillomas were present in similar sites as carcinoma in 50 per cent of the animals and urinary bladder papillomas were identified in 11 percent of the animals.

On the basis of a 15 month study, it was calculated that the mortality rate due to upper alimentary squamous cell carcinoma was a minimum of 2.5 per cent of the cattle population of Nasampolai valley per year. This high frequency which did not appear to occur elsewhere in the Narok district was attributed to the unique grazing patterns of the cattle. Throughout the valleys of the Narok district the cattle graze almost exclusively on the moorland type vegetation of the valley floors, whereas in Nasampolai population pressures necessitated the frequent use, particularly during periods of drought, of clearings in the dense bamboo forest on the walls of the valley. The apparent association between forest grazing and the occurrence of upper alimentary squamous cell carcinoma in the cattle was further indicated when squamous cell carcinoma of the oesophagus and proventricular (oesophageal) region of the stomach was found in two giant forest hogs

(Hylochoerus meinertzhageni, Thomas), a species which lives and grazes exclusively in the forest areas. Consequently Plowright and others (1971) hypothesised that a carcinogen or carcinogen precursor present in a forest plant or plants was involved in the aetiology of the neoplasm. Although the flora included bracken fern, its possible role was excluded on the grounds that it was not considered to be an item of cattle forage and the owners of the cattle did not recognise the clinical syndrome associated with urinary bladder neoplasia which occurs on bracken infested pastures, despite the fact that the authors themselves had identified such neoplasms. The only alternative aetiology which has been suggested was made by Magee and Barnes (1967) who, when commenting on the initial investigations of Plowright (1955), speculated that a nitroso-compound could be present in a plant consumed by the cattle, but there has been no subsequent evidence that this is the case.

In Brazil, a high frequency of upper alimentary squamous cell carcinoma is recognised in well defined areas which extend through several provinces in the south eastern region of the country (Dobereiner and others, 1967; Tokarnia and others, 1969). Although no incidence rates are available, the neoplasm is so common within these areas that local vernacular names such as 'crava da goela' and 'verrugo da goela' are used by farmers to describe the condition which affects a wide variety of breeds of cattle of both sexes aged between five and 15 years. The squamous carcinomas, which usually arise as multiple primary foci, involve one or more of the following

sites; the base of the tongue, palate, pharynx, oesophagus, cardia and anterior dorsal sac of the rumen. Ninety per cent of the affected animals also have papillomas situated in the same sites in the upper alimentary tract and approximately 40 per cent are simultaneously affected by various urinary bladder neoplasms including haemangiomas and adenocarcinomas.

As bovine urinary bladder neoplasia is common in south eastern Brazil, where it occurs in cattle grazed on bracken infested pastures, the simultaneous presence of upper alimentary squamous cell carcinoma and urinary bladder neoplasia in a large number of animals suggested that bracken fern could also be involved in the aetiology of upper alimentary squamous cell carcinoma, and investigations of the origins of affected animals indicate that the neoplasm is confined to areas where there is bracken infestation (Dobereiner and others, 1967; Campos Neto and others, 1975).

However, it has been suggested that a virus may also be implicated in the aetiology of upper alimentary squamous cell carcinoma . Several authors have observed that a high proportion of animals affected by upper alimentary squamous cell carcinoma also have upper alimentary papillomas and Trotter (1911) and Plowright (1955) suggested that the carcinoma is the result of malignant transformation of these papillomas. The recent identification of a papilloma virus in upper alimentary papillomas by Jarrett, Murphy, O'Neill and Laird,(1978) has led to the proposal that the development of carcinoma may be

the result of an interaction between the carcinogen(s) present in bracken fern and the papilloma virus responsible for upper alimentary papillomas (Jarrett, McNeil, Grimshaw, Selman and McIntyre, 1978).

Upper Alimentary Papillomas

Papillomas are considered to be the most common neoplasm of the oesophagus in cattle (Cotchin 1957; Nieberle and Cohrs, 1967) and the latter authors state that in animals affected by oesophageal papillomas, similar lesions are often present in the mouth and pharynx. Other sites in the upper alimentary tract in which papillomas can be found include the oesophageal groove and rumen (Moulton, 1961; Smith and Jones, 1961) and Nieberle and Cohrs (1967) and Jubb and Kennedy (1976) record that papillomas can occur at any site in the forestomachs. In addition, several authors (Trotter, 1911; Plowright, 1955; Dobereiner and others, 1967) have reported that upper alimentary papillomas are commonly present in animals affected by upper alimentary squamous cell carcinoma and have suggested that the papillomas may be involved in the aetiology of the carcinoma.

However despite the apparent frequency with which papillomas occur, there is very little known regarding their epidemiology, aetiology or significance.

Two surveys have been carried out, both in abbatoirs, to determine the prevalence of upper alimentary papillomas. Thorsen, Cooper and Warwick (1974) undertook a survey of oesophageal papillomas at a Kenya Meat Commission abbatoir near Nairobi as an extension of the investigation of the high incidence area of upper alimentary squamous cell carcinoma described by Plowright and others (1971). Two macroscopic types of papillomas were identified; a small

flattened type with a sponge-like surface and a larger type measuring up to 1cm diameter and 8mm high which was often elongated with finger-like projections protruding from the apex into the lumen of the oesophagus. The prevalence of these papillomas in the 752 oesophagi examined was 6.1 per cent but in most cases less than three papillomas were found and the maximum number present in any individual oesophagus was 21. These small numbers of papillomas were noted to be in marked contrast with the large numbers, occasionally exceeding 100, which could be found in animals in the high incidence area of upper alimentary squamous cell carcinoma.

Thorsen and others (1974) give no indication of the age prevalence of oesophageal papillomas in their survey and apart from recording that the animals examined, "originated from widely separated geographical areas with varying climatic conditions and vegetation", the authors give no indication of any geographical variation in the occurrence of upper alimentary papillomas.

Jarrett and others (1978b) conducted an abbatoir survey of upper alimentary papillomas at an abbatoir in Glasgow, Scotland. Three distinct lesions were described of which two were considered to be squamous papillomas and the third, which was only found in the oesophagus, a fibroma or fibropapilloma. The first type of squamous papillomas was composed of a number of fronds or subunits, each of which terminated in a keratinised tip. In the oesophagus, these lesions were usually acuminate in shape but at sites of friction, such as the tongue or palate, had a blunted appearance. The second type of squamous

papilloma was a sessile growth with a flattened top and no obvious subunit division. The fibroma or fibropapilloma was also a sessile lesion which was morphologically similar to the cutaneous fibroma or fibropapilloma found in cattle.

One of more of these lesions were present in 19 per cent of 2746 animals in which the entire upper alimentary tract was examined and 8.2 per cent of 5000 cases in which only the oesophagus was examined. The age range of affected animals was seven months to 16 years and no significant difference could be demonstrated in either the prevalence or the numbers of upper alimentary papillomas amongst animals of differing age groups. There was also no evidence of an unequal sex distribution. The numbers of papillomas present in individual animals were usually very few and only eight per cent of 639 affected animals examined in detail had more than three papillomas. In addition, in 95 per cent of these animals the papillomas were confined to one site in the upper alimentary tract. Jarrett and others (1978b) draw attention to the marked contrast between the small numbers of papillomas found in animals in their abbatoir survey compared with the much larger numbers present in a multiplicity of sites in animals affected by upper alimentary squamous cell carcinoma.

An attempt was also made by Jarrett and others (1978b) to trace the farms of origin of animals which were found to have papillomas in their abbatoir survey. This was accomplished in 66 cases, the vast majority of which originated on beef or dairy lowland farms. It is also recorded that very few animals originated on bracken infested upland farms.

Although it has long been suggested that upper alimentary papillomas have an infective aetiology (Kitt, 1921; Nieberle and Cohrs, 1949) the presence of virus in these lesions has only recently been demonstrated. Jarrett and others (1978b)reported the presence of type A intranuclear inclusion bodies in 13 per cent of 78 randomly selected upper alimentary papillomas and, on electron microscopy, the presence of large intranuclear crystalloid assays of virus particles. The structure of the virus was considered to be identical to that of bovine cutaneous papilloma virus (Jarrett and others, 1978b)and when injected into the epithelium of the mouth and skin gave rise to papillomas (Jarrett, 1978).

Intestinal Adenocarcinoma

Intestinal adenocarcinoma is a rarely reported bovine neoplasm and Lingeman and Garner (1972) in their comparative study of the tumour in animals and man could only find published data on 36 cases in the world literature, many of which were inadequately described. Subsequently further examples have been described by Damodaran and Parthasarathy (1973) and Vitovec (1976) who confirm that the neoplasm is usually situated in the jejunum, and is only rarely found in the remainder of the small intestine or large intestine. Nothing is known regarding the epidemiology of intestinal adenocarcinoma in cattle other than it is a neoplasm which is confined to adult or aged animals (Vitovec, 1976) and accounts for between less than one per cent (Anderson and others, 1969) to over five per cent (Misdorp, 1967) of malignant neoplasms identified in various surveys.

Urinary Bladder Neoplasia

Urinary bladder neoplasms have been recognised as the cause of enzootic bovine haematuria in many countries throughout the world (Figure 29).

Clinical disease is rarely observed in animals under two years of age (Moussu, 1904; Georgiev, 1957; Pamukcu, 1955) and usually animals are at least three years or older before they come affected (Bull and others, 1932; Datta, 1953; Dobereiner and others, 1967; Smith and Beatson 1970) The peak age incidence most commonly recorded is between four and six years (Hadwen, 1917; Bull and others, 1932; Datta, 1953; Pamukcu, 1955; Beran, 1966) but it is generally agreed that enzootic haematuria can occur at any age over three years and cases are frequently recorded in animals aged over six years (Kalkus, 1913; Roberts, 1923; Craig, 1930; Bankier, 1943; Pamukcu, 1955). On individual farms the ages at which animals are affected tends to vary and occasionally younger animals may be affected whereas older animals are not (Bankier, 1943).

Most authors record that male and female cattle are equally affected (Moussu, 1904; Hadwen, 1917; Bankier, 1943; Pamukcu, 1955; Georgiev, 1957; Butozan and Mihajlovic 1959 Beran, 1966). However Roberts (1923) and Craig (1930) state that in Great Britain only females are affected.

Enzootic haematuria has been reported in a wide variety of breeds of cattle (Bankier, 1943; Georgiev, 1957) and also occurs in water buffalo in Turkey (Pamukcu, 1955 and 1957) Taiwan (Miyamoto, 1927; Goto and others, 1954)

FIGURE 29 The Reported World Distribution of High Incidence Areas of Urinary Bladder Neoplasia in Cattle.

and Indonesia (Ressang and Sikar, 1960). However, Beran (1966) stated that water buffalo, which grazed with cattle which became affected, did not appear to develop the disease, and suggested that this difference could be due to the more selective grazing habits of the water buffalo. Seasonal variations with peak incidences in the late summer, autumn and early winter have been reported (Craig, 1930; Burnett, 1937; Butozan and Mihajlovic, 1959). However the majority of author agree that this does not occur and Smith and Beatson (1970) attribute apparent seasonal variations to the observation of affected animals at peak handling times.

In countries where urinary bladder neoplasia is commonly recognised the malignancies tends to be confined to cattle grazing in specific enzootic areas and although many such areas are relatively small, some are extremely large and extend over thousands of square miles. The largest enzootic area is probably in northern Turkey where a high prevalence of bovine urinary bladder neoplasia is recognised in a belt which extends for over 600 miles along the southern coast of the Black Sea (Pamukcu, 1963).

Usually, enzootic areas are confined to upland or mountainous regions (Roberts, 1923; Butozan and Mihajlovic, 1959; Pamukcu, 1963), but, occasionally, they occur on low lying ground as has been reported in France and on the western seaboard of Canada (Hutyra, Marek and Manninger, 1949; Beran, 1966). Regardless of these differences in altitude, the poor quality of pasture is an almost constant feature. In many countries, including

Canada (Kalkus, 1913; Hadwen, 1917), Ireland (Craig, 1930), Rumania (Martincic, 1955), and Yugoslavia (Gregorovic, Jazbec, Senk and Skusek, 1970) the pastures have been described as poor and badly drained, uncultivated, undercultivated or falling into neglect. Enzootic areas also occur on heavily forested land in India (Datta, 1953), Turkey (Pamukcu, 1963) and England (Hall Mashter, 1933) and recently cleared forest which is only partially cultivated in Canada (Bankier, 1943).

However one feature of enzootic areas, which appears to be almost universal, is the presence of bracken ferm (Pteridium aquilinum) and only one report categorically states that bracken fern does not grow in an enzootic area (Mugera and Nderito, 1969).

The prevalence of the disease varies considerably within enzootic areas, and only some of the farms within an area are affected (Bankier, 1943; Pamukcu, 1963). It is not unusual for a farm which suffers heavy losses to be adjacent to another on which the disease does not appear to occur (Craig, 1930; Bull and others, 1932; Burnett, 1937; Pamukcu, 1963). However, none of these authors consider the possible variations between farms in management systems, particularly concerning the grazing patterns and life expectancies of the cattle. Generally the disease is sporadic but in some herds a large proportion of the animals may be affected either simultaneously or over a period of months or years (Kalkus, 1913; Craig, 1930; Bull and others, 1932; Bankier, 1943; Datta, 1953; Smith and Beatson, 1970).

The 'incidence' of haematuria within enzootic areas has been estimated up to and over 15 per cent (Kalkus, 1913; Pamukcu, 1955, 1957; Butozan and Mihajlovic, 1959; Georgiev, Labor, Stoyanor, Dankov, Krustev and Nikolaev, 1962) and within severely affected herds up to 30 per cent (Bankier, 1943; Martincic, 1955), 50 per cent (Kalkus, 1913) or even 90 per cent (Georgiev, 1957). In New Zealand, Smith and Beatson (1970) recorded that in some herds, eventually all of the older animals become affected. The period of exposure in an enzootic area for development of the lesions producing clinical haematuria is unknown, but when animals are moved into an enzootic area from a haematuria free area the minimum time for signs of the disease to appear is approximately two to three years (Bull and others, 1932; Pamukcu, 1955; Rosenberger, 1971). Similarly, when animals are moved out of an enzootic area they may develop the disease up to two to three years later (Bankier, 1943), and Georgiev (1960) has described a case in which haematuria did not develop until six years later.

Lymphosarcoma

Lymphosarcoma (Syn. malignant lymphoma, bovine leukosis) has been recognised as a disease of cattle for over 100 years (Bollinger 1874, cited by Olsen and

Baumgartener, 1975) and, during the last 50 years, has attracted considerable attention as the result of its distinct epidemiological characteristics. In many countries the disease assumes an enzootic form in which a high incidence of the disease is recognised in adult cattle, multiple cases occur within individual herds and there is evidence of spread of the disease within and between herds. In addition, a proportion of apparently normal cattle within herds in which cases occur may be found to have abnormally high lymphocyte counts i.e. lymphocytosis. This form of lymphosarcoma is generally known as enzootic bovine leukosis. Many of the early descriptions of enzootic bovine leukosis were made in Germany where, prior to World War II, a high incidence of leukosis was recognised in the north eastern region (Dobberstein and Paarman, 1934; Schottler and Schottler, 1934). However, during and after the war, movement of cattle gradually spread the disease westwards (Gotze, Rosenberger and Ziegenhagen, 1956) leading to the suggestion that it was transmissible. Similarly, in Sweden, a high incidence of leukosis was recognised in adult cattle in the immediate post war period (Hansen and Winquist, 1961) but in this country much of the spread of the disease was attributed to transmission by a whole blood vaccine against babesiosis (Hjärre, 1958; Olson, 1961).

Enzootic bovine leukosis has also been recorded and studied in detail in many other countries including Denmark (Bendixen, 1965) and the United States (Marshak and others, 1962; Theilen, Appleman and Wixom, 1963).

Although enzootic bovine leukosis has accounted for most of the interest shown in lymphosarcoma it has been demonstrated that a second epidemiological form of the disease exists. In Denmark, Bendixen (1963) reported that in addition to enzootic bovine leukosis which was only found in adult animals aged four years and older and presented clinically as multicentric lymphosarcoma there was a sporadic form which occurred in both adult and immature animals. In this form of the disease only one case was found in any individual herd, there was no contact with multiple case herds and none of the other animals in the herd had evidence of lymphocytosis. In addition, adult animals with sporadic leukosis presented with skin lymphosarcoma and immature animals had either multicentric or thymic lymphosarcoma. Subsequently, immature animals with sporadic bovine leukosis have been described both in countries in which enzootic bovine leukosis is recognised e.g. the United States (Theilen and Dungworth, 1965; Dungworth and others, 1964) and in countries in which the enzootic form of the disease is not thought to occur e.g. New Zealand (Shortridge and Cordes, 1971). However, other than in Denmark, the skin form in adults has rarely been recorded.

As a result of the epidemiological evidence of the transmissible nature of enzootic bovine leukosis various attempts were made to demonstrate a viral aetiology by transmission experiments and virus isolation (Dutcher, Szekely, Larkin, Coriell and Marshak, 1963; McKercher, Wada, Straub and Theilen, 1963). However these attempts were unsuccessful or inconclusive until Miller, Miller, Olson and Gilette (1969) demonstrated the presence of virus-like particles in stimulated lymphocyte cultures. Subsequently it was shown that the virus responsible for enzootic bovine leukosis is a member of the retravirus family (Burny, Cleuter, Dekegel, Glysdael, Kettmann, Mammerickx and Portetelle, 1976) which is exogenous and is not transmitted through the germ cell line (Callahan, Lieber, Todaro, Graves and Ferrer, 1976).

A range of serological tests have now been developed for the detection of specific antibodies against enzootic bovine leukosis virus antigens (Mussgay and Kaaden, 1978) including an agar gel immunodiffusion test based on a glycoprotein antigen (Straub, 1978) which has been adopted as an official test within the European Economic Community. These serological tests have thus enabled identification of infected animals prior to the development of clinical disease and in some countries control programmes have recently been instituted.

In contrast the aetiology of the sporadic form of lymphosarcoma remains totally unknown.

Until 1978, it was thought that only the sporadic form of lymphosarcoma was present in the United Kingdom. Surveys performed by Cotchin (1960) and Anderson and others (1969) served to confirm this point of view in that the majority of animals with lymphosarcoma which these authors examined were immature. Anderson and Jarrett (1968) stated that no multiple case herds with affected adult animals had been reported in the United Kingdom and that only three herds had ever been identified in which more than one case of lymphosarcoma had occurred in immature animals. Subsequently, Chasey, Wibberley, Markson and Roberts (1978) have demonstrated the presence of enzootic leukosis virus infection in British cattle which is thought to have been introduced in imported Canadian cattle approximately six years earlier. However, to date, only one confirmed clinical case of enzootic bovine leukosis has been reported in the United Kingdom (Grimshaw, Wiseman, Petrie, Selman, Gibbs and Thompson, 1980).

The Toxicity of Bracken Fern

Attention has been drawn to the association between neoplasia, particularly that of the urinary bladder in cattle, and bracken fern (Pteridium aquilinum) by numerous authors

and subsequently the toxicity of this plant has been investigated in detail. (Figure 30). It has been demonstrated that the plant contains two or more toxic compounds which exert anti-thiamine, acute radiomimetic or carcinogenic effects depending on the quantity and to which species the plant is administered.

The anti-thiamine property of bracken fern is due to the presence of a powerful thiaminase which tends to be concentrated in the rhizomes (Evans, Jones and Evans, 1950) and can induce thiamine deficiency (avitaminosis B_1) in simple stomached animals including the horse (Evans, Evans and Roberts, 1951), the pig (Evans, Humphreys, Goulden, Thomas and Evans, 1963), the rat (Weswig, Freed and Haag, 1946; Evans and Evans, 1949), and the pigeon (Jones, 1952). In contrast, ruminants seldom succumb to the effects of the thiaminase present in bracken fern. This is due to the synthesis of B group vitamins, including thiamine, by the rumen microflora which, when autolysed in the abomasum whose low pH is unfavourable for thiaminase activity, liberates absorbable thiamine (Evans, Evans, Thomas, Watkins and Chamberlain, 1958). However, despite this mechanism, bracken fern induced thiamine deficiency resulting in cerebrocortical necrosis has been produced experimentally in sheep (Evans, Evans, Humphreys, Lewin, Davies and Axford, 1975).

FIGURE 30 Bracken Fern (Pteridium aquilinum)

In contrast to thiaminase activity which exists in a variety of plants including horsetail (Equisetum) and cockscomb (Celosia cristata), acute radiomimetic properties appear to be confined to bracken and one other fern, Cheilanthes sieberi (rock fern or mulga) which is found in Australia and New Zealand (Clark and Dimmock, 1971; McKenzie, 1978). Cattle are particularly susceptible to the acute radiomimetic properties of bracken fern and the consumption of large quantities produces the rapidly fatal syndrome known as 'acute bracken poisoning' which is characterised clinically by widespread mucosal haemorrhage, marked pyrexia and profound dullness (Figures 31 & 32). The interval between the onset of consumption of bracken fern and the development of clinical evidence of disease depends on the amount consumed and the toxicity of the plant which varies during the growing season. The toxicity of the fronds is greatest in the early stages of growth and thereafter declines, but even the dead fronds retain considerable toxicity. Although acute bracken poisoning usually becomes evident during a period of continued consumption lasting at least one month, overt clinical signs can develop up to six weeks after consumption has ceased. These clinical signs are the consequence of the effects of a toxin, the structure of which is unknown (vide infra), in sites of rapid cell division (Naftalin and Cushnie, 1954a; 1954b; Evans, Evans and Hughes, 1954). Bone marrow aplasia is induced and causes the development of a marked leukopaenia, particularly neutropaenia, and thrombocytopaenia which results in a prolonged bleeding time. There is increased

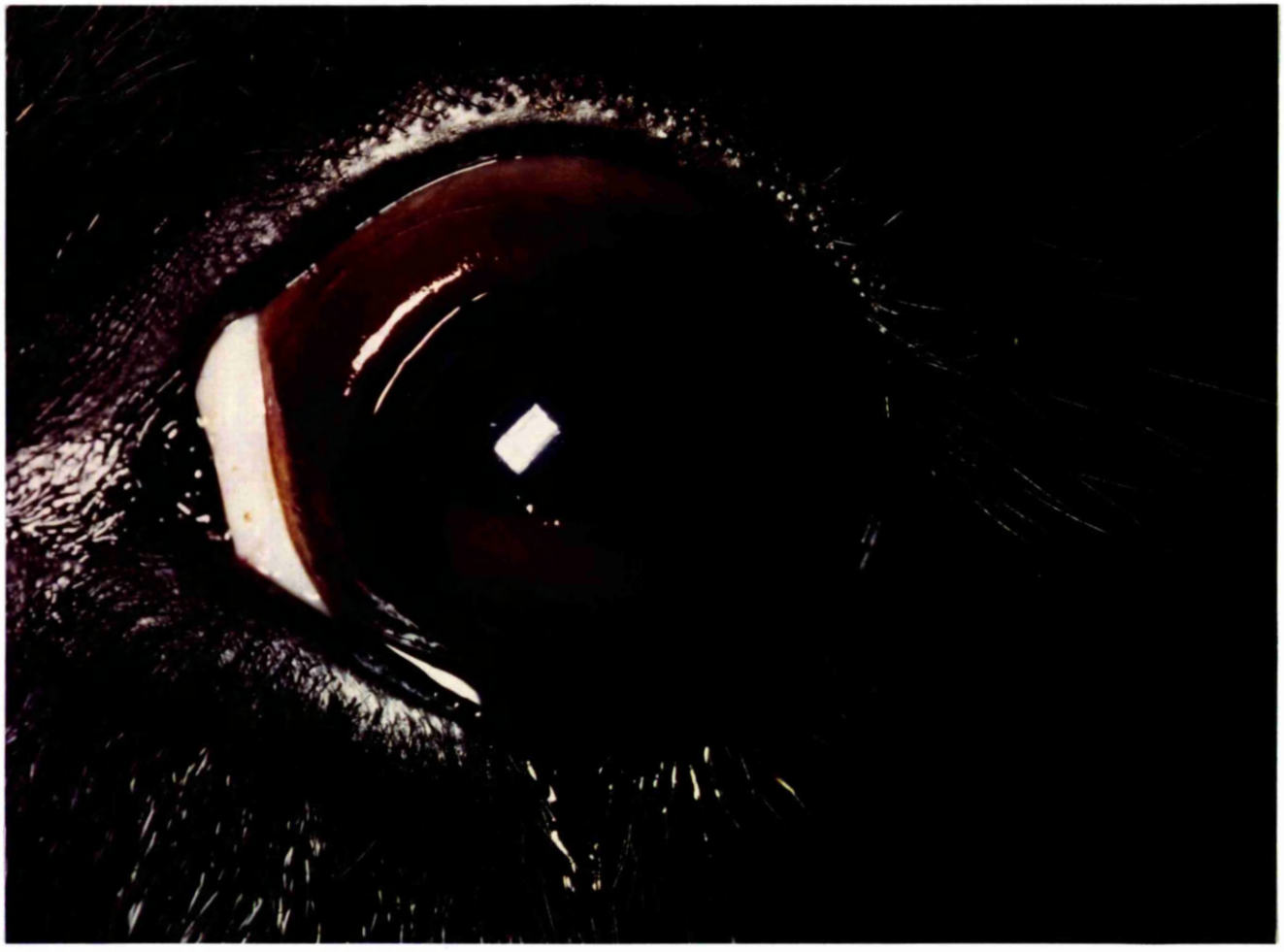


FIGURE 31 Acute Bracken Poisoning : Intraocular haemorrhage and petechiation and marked pallor of the nictitating membrane

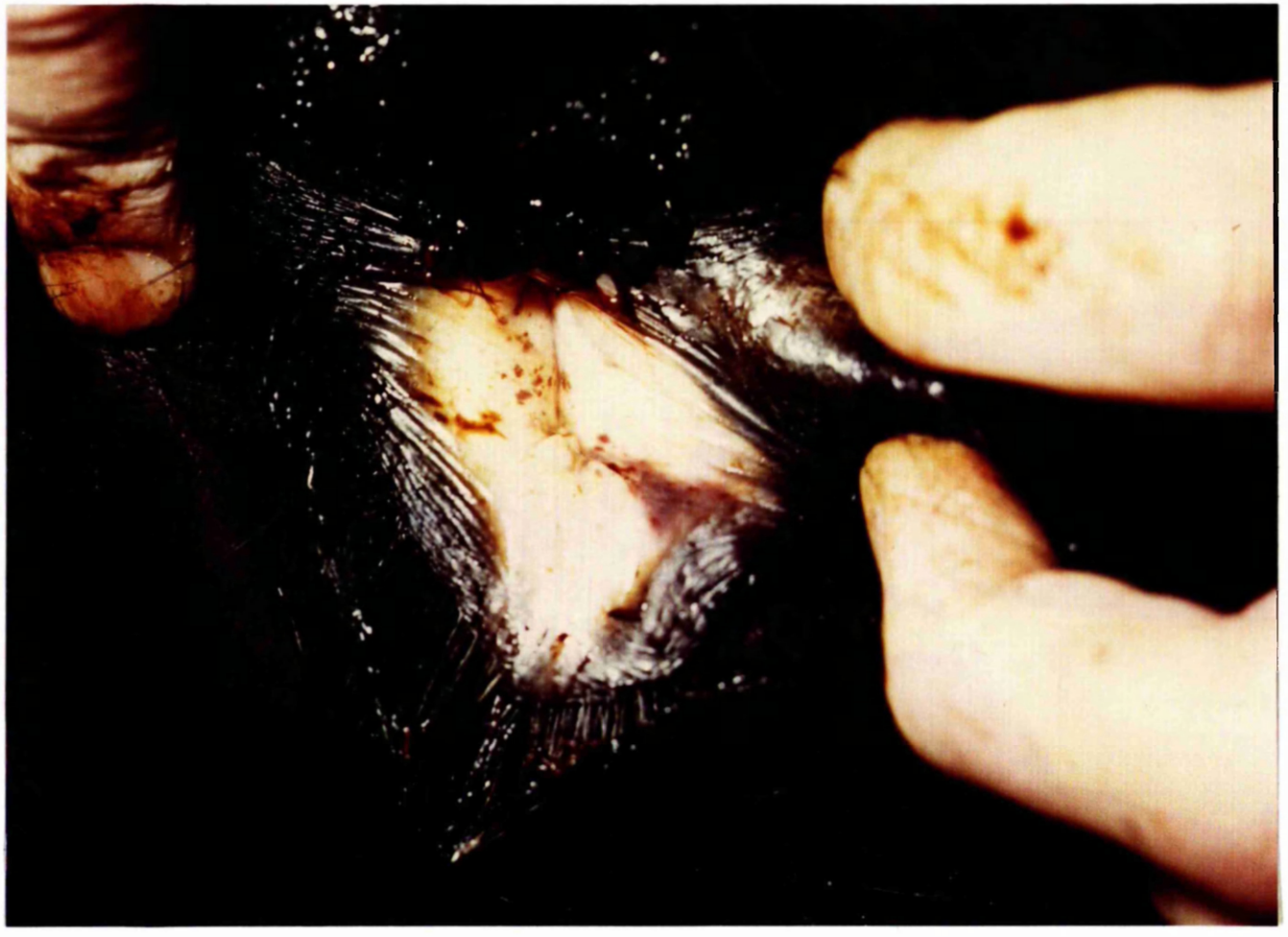


FIGURE 32 Acute Bracken Poisoning : Petechial and ecchymotic haemorrhages of the vulva with marked pallor of the vulval mucosa. Note also the presence of melaena at the anal orifice

capillary fragility and defective clot retraction which contribute to the mucosal haemorrhage. The severe ulceration of the intestinal mucosa which is seen in cattle with acute bracken poisoning has also been attributed to the effects of the toxin on rapidly dividing cells. Sheep can also succumb to acute bracken poisoning (Moon and Raafat, 1951) but, compared to cattle, they appear to be much less susceptible to the effects of the toxin (Moon and McKeand, 1953).

Evans, Thomas, Evans and Edwards (1958) and Heath and Wood (1958) commented on the similarity between acute bracken poisoning and the changes which occur on exposure to ionising radiation,and subsequent experimental irradiation of cattle by Schultze, Perman, Mizuno, Bates, Sautter, Isbin and Lokens (1959), Brown, Thomas, Jones, Cross and Sasmore (1961), and Brown (1962) has demonstrated the almost identical nature of the two conditions.

The carcinogenic activity of bracken fern has been demonstrated in a variety of species of animals in which long term feeding of the plant results in the induction of benign and malignant neoplasms. The most extensive trials have been performed in rats in which intestinal and urinary bladder neoplasms can be induced. Evans and Mason (1965) fed pellets which contained one third dried, milled bracken fronds by weight to 40 seven week old non-inbred Hooded Lister rats for nine weeks. During the period of bracken consumption the rats were given supplementary thiamine on three occasions by subcutaneous injection to counteract the effects of the thiaminase present in the

bracken. Forty control animals were similarly administered thiamine. Between seven months and one year after the onset of bracken consumption, all the experimental rats had developed multiple adenocarcinomas of the intestine, which were concentrated in the ileal region, whereas no tumours were found in the control animals.

These results were confirmed by Pamukcu and Price (1969), Pamukcu, Yalciner, Price and Bryan (1970), Hirono, Shibuya, Fushimi and Haga (1970) and Hirono, Fushimi, Mori, Miwa and Haga (1973) using four to seven week old Albino and A.C.I. rats fed bracken in their diets for periods ranging between 16 and 48 weeks. In addition to adenocarcinomas of the intestine, intestinal adenomas were frequently present and Hirono and others (1970) identified intestinal fibrosarcomas in 50 per cent of their animals. Urinary bladder neoplasms were also found in a proportion of the bracken-fed rats in each of these experiments, the types identified being papillomas, squamous and transitional cell carcinomas and adenocarcinomas. Pamukcu and Price (1969) and Pamukcu and others (1970), who fed Albino rats bracken in their diet continuously until they died between 24 and 48 weeks later, reported that 81 and 62 per cent of the rats respectively had urinary bladder tumours. However Hirono and others (1970) and Hirono and others (1973) only found urinary bladder neoplasms in four and six per cent respectively, one animal in each case, of their ACI rats fed bracken for 16 weeks.

The reasons for the variation in the types and sites of neoplasms which can be induced in bracken fed rats has not been adequately explained. Amongst the differences in experimental procedure which could be responsible are the breeds of rats utilised, the length of time of bracken ingestion, the quantity and toxicity of the bracken ingested and whether or not supplementary thiamine was administered. It would appear from some of the experimental results that the longer bracken is fed, the more likely the development of urinary bladder neoplasms. However, the trial conducted by Schacham, Philp and Gowdey (1970), in which rats were fed bracken for a similar length of time as in the experiments performed by Pamukcu and Price (1969) and Pamukcu and others (1970), did not result in the development of urinary bladder tumours, although all the rats did become affected by intestinal adenocarcinoma. Pamukcu and others (1970) reported that the administration of parenteral thiamine to bracken fed rats increased the incidence of urinary bladder neoplasms from nine per cent in unsupplemented animals to 62 per cent in those which received parenteral thiamine. Unfortunately the relative importance of the factors which could affect the induction of urinary bladder neoplasms cannot be assessed from the published information.

In contrast, the induction of intestinal adenocarcinoma appears to be readily repeatable, although an age susceptibility to the carcinogenic effects of bracken has been demonstrated, in that older rats are less susceptible (Evans and Widdop, 1966).

The administration of bracken fern to mice has demonstrated its carcinogenic properties in this species, but, as in the rat, there has been considerable variation in the type and sites of neoplasms induced. Evans and Widdop (1966) and Evans (1968) record that non-inbred, six week old, female Swiss white mice continuously fed pellets containing one third bracken for five weeks, all developed pulmonary adenomas and died within 19 months. However, in later experiments, using either whole bracken or ethanol extracts of bracken administered in the feed, or by stomach intubation or intraperitoneal infection, Evans, Barber, Jones and Leach (1969) produced a wide range of pulmonary, hepatic, and haemopoietic tumours, including lymphatic leukaemia. Pamukcu, Erturk, Price and Bryan (1972) also observed the development of lymphatic leukaemia in all six week old 'spontaneous leukaemia free' Swiss mice fed 33 per cent bracken pellets on alternate weeks for 60 weeks, but found pulmonary adenomas or adenocarcinomas in only 15 per cent. In addition, the induction of squamous papillomas and carcinomas of the non-glandular area of the mouse stomach and carcinoma, in situ, of the pyloric region by single oral or parenteral administration of bracken extracts has been recorded by Evans (1972), Jones (1974) and Evans (1976). These authors have also reported the development of adenocarcinoma of the pyloric region in a few cases.

The carcinogenic properties of bracken have been further demonstrated in other laboratory species including guinea pigs and Japanese quail. Evans (1968) fed six week

old guinea pigs fresh bracken fronds ad lib for 11 weeks as a supplement to their normal diet. Between 17 and 28 months later some of the guinea pigs exhibited chronic intermittent haematuria and at necropsy, 30 months from the outset of the experiment, all were found to have developed urinary bladder tumours including papillomas, papillary carcinomas and transitional cell carcinomas. One guinea pig which died after only 23 months had an adenocarcinoma of the jejunum but no bladder tumours. The production of urinary bladder tumours in guinea pigs fed bracken has subsequently been confirmed by Bryan (1977).

Eighty per cent of Japanese quail (Cotornix cotornix japonica) fed on an ethanol extract of dried bracken mixed with their normal diet for five months after hatching developed intestinal adenocarcinoma (Evans, Widdop and Barber, 1967). The carcinomas were predominantly situated in the caecae but were also found in the colon and distal ileum.

Several attempts have been made to demonstrate the carcinogenic properties of bracken in cattle. Rosenberger and Heeschen (1960) fed fresh and dried bracken to eight cattle which thereafter developed microhaematuria within 8½ to 13 months of the onset of bracken supplementation of their diets and overt haematuria within 10½ to 16 months. Seven of the cattle eventually died of acute bracken poisoning but all were stated to exhibit the pathological changes typical of naturally occurring enzootic haematuria. However, the descriptions of the lesions observed in the bladder are extremely poor, leaving in

doubt whether or not they were actually neoplastic in nature. Pamukcu (1963) performed a similar experiment on eight cattle, but once again this was terminated prematurely by acute bracken poisoning. During the experiment four animals exhibited intermittent microhaematuria and one, which had been fed bracken for 360 days, was found, at post mortem, to have a small papilloma on the mucosa of the bladder. However, by prolonging the feeding of bracken to between two and three years, Sofrenovic, Stamatovic and Bratanovic (1965) produced haemangiomas and papillomas of the bladder in four of their five experimental cattle.

Thereafter, Pamukcu, Goskay and Price (1967) demonstrated that bracken is capable of producing both benign and malignant neoplasms of the bovine urinary bladder, provided consumption is continued over a prolonged period. A variety of urinary bladder neoplasms were produced in ten cattle which were fed bracken for between 276 and 1192 days. Eight of the animals developed microhaematuria and, or macroscopic haematuria with remissions typical of the disease in the field. Similarly, Price and Pamukcu (1968) reported the induction of various benign and malignant bladder neoplasms with clinically evident haematuria in six cattle fed bracken for between 510 and 1920 days. The tumours produced in these two experiments were haemangiomas, papillomas, a fibroma, transitional and squamous cell carcinomas, haemangioendotheliomas and a mucous adenocarcinoma which comprise all the most commonly recognised tumours of the field disease.

At present, it is unknown whether the acute radiomimetic and carcinogenic properties of bracken are due to a single substance in the plant, although, from the available evidence this would seem probable. Despite the considerable amount of effort which has been expended, there has been little success in the identification of the toxin or toxins involved.

Hot ethanol extracts of bracken which were subjected to additional solvent purification were shown to have retained both the acute radiomimetic and carcinogenic properties of the bracken (Evans and Widdop, 1966; Widdop, 1967). When column and thin layer chromatography was performed on this extract, a fraction was isolated which was acutely toxic mutagenic and carcinogenic in mice (Evans and others, 1969; Leach, Barber, Evans and Evans, 1971). Osman (1974) identified the main compound in this fraction as shikimic acid, 3,4,5 trihyroxy-1-cyclohexene-1-carboxylic acid. However, although it has been shown that shikimic acid has mutagenic and carcinogenic activity in mice (Evans and Osman, 1974) and that it is teratogenic in mice and quail (O'Donovan, Brewster and Jones, 1977; Prorok, 1978; Evans, 1979), Hirono, Fushimi and Matsubara (1977) were unable to demonstrate any carcinogenic activity in rats. Similarly, a tannin isolated from bracken by Wang, Chiu, Pamukcu and Bryan (1976), is acutely toxic to mice and produces carcinomas in the bladder of the mouse when implanted in cholesterol based pellets, but subsequent studies by Pamukcu, Wang, Hatcher and Bryan (1980) failed to demonstrate carcinogenicity on oral administration to rats whereas a

tannin-free fraction of bracken fern was shown to cause intestinal adenoma and adenocarcinoma.

Other studies directed at the isolation of carcinogenic compounds from bracken have also been carried out by Yoshihira, Fukuoka, Kuroyanagi, Natori, Imeda, Morohoshi, Enomoto and Saito (1978) who isolated twenty sesquiterpenes with a 1-indanone nucleus which were identified as pterosins and their glycosides, pterosides. However none could be shown to be either mutagenic or carcinogenic. Fukuoka, Kuroyanagi, Yoshihira, Natori, Nagao, Takahashi and Sugimura (1978) identified various flavonoids in bracken fern, including quercetin and kaempferol and their glycosides isoquercitrin, rutin and astragalin. On examination of extracts containing these compounds it was found that kaempferol exhibited particular mutagenicity to Salmonella typhimurium strains TA 100, TA 98 and S - 9 mix. Subsequently, Pamukcu, Yalciner, Hatcher and Bryan (1980) investigated the carcinogenicity of quercetin, its structural analogue kaempferol, and rutin, and reported that quercetin produced carcinogenic changes in the intestinal and urinary bladder epithelium of rats identical to those caused by bracken fern. In contrast, Hirono, Ueno, Hosaka, Takanashi, Matsushima, Sugimura and Natori (1981), who fed experimental diets containing quercetin or rutin to an inbred strain of ACI rats, known to be highly susceptible to the carcinogenic properties of bracken fern, were unable to find any significant difference in the incidence of

neoplasia in the experimental groups compared with the negative controls. The only explanation suggested for the disparity between their results and those of Pamukcu and others (1980) was that a different strain of rat was used, the latter authors having employed Norwegian rats.

Contradictions similar to that described immediately above exist throughout the literature relating to the carcinogenicity of bracken fern. The failure of the various groups working in this area to standardise techniques and criteria of interpretation of results can only have contributed to the continued lack of success in the identification of its toxic components.

AN EPIDEMIOLOGICAL STUDY OF BOVINE NEOPLASIA WITH PARTICULAR REFERENCE TO ALIMENTARY AND URINARY BLADDER NEOPLASMS AND THEIR RELATIONSHIP WITH BRACKEN FERN (PTERIDIUM AQUILINUM)

INTRODUCTION

Epidemiology may be defined as the study of a disease or physiological condition in (human) populations and of the factors which influence its distribution (Lilienfeld, 1976). In respect of neoplasia in humans, numerous epidemiological studies have been undertaken in attempts to identify the aetiological factor(s) responsible. A number of these studies have been successful, e.g. the identification of a direct association between urinary bladder neoplasia and exposure to specific chemicals used in industry (Case, Hosker, McDonald and Pearson, 1954), but in the vast majority of cases there has been no conclusive demonstration of a specific aetiology, despite the many associations which have been demonstrated between environmental and sociological factors and different forms of neoplasia.

There have been few detailed epidemiological studies of neoplasia in animals but their value is exemplified by the investigations of Bendixen and other workers, reviewed by Bendixen (1965), which suggested that certain forms of bovine lymphosarcoma were transmissible and led to the identification of the viral aetiology of enzootic bovine leukosis (vide supra).

There is no doubt that epidemiological studies of neoplasma in animals can make a substantial contribution to

the understanding of the mechanisms of induction of neoplasia, not only in animals but also in humans, and to this end the following chapter is devoted to a study of the epidemiology of bovine neoplasia.

SECTION I

THE AGE, BREED AND SEX DISTRIBUTION OF MALIGNANT NEOPLASMS AND ASSOCIATED BENIGN NEOPLASMS

INTRODUCTION

Although it is widely recognised that the frequency of malignant neoplasia in cattle increases with age, there is a dearth of information in relation to the age prevalence of specific neoplasms. However a notable exception to this generalisation is lymphosarcoma which has been studied in detail and has been shown primarily to affect immature cattle in some countries e.g. the United Kingdom and New Zealand (Cotchin 1960; Anderson and others, 1969; Shortridge and Cordes, 1971) whereas in other countries e.g. the United States and the Netherlands, it is predominantly observed in adult cattle (Monlux and others, 1956; Misdorp, 1967).

Variations in the frequency of neoplasia amongst different breeds or types of cattle have occasionally been recognised. For example, in the United States, the most common malignancy in dairy type animals is lymphosarcoma whereas in beef type animals it is ocular squamous cell carcinoma (Brandley and Migaki, 1956). This latter neoplasm has also been associated specifically with the Hereford breed, not only in the United States, where it is the major beef breed (Priester and Mantel, 1971), but also in other countries, including New Zealand, where the Aberdeen Angus breed predominates (Shortridge and Cordes, 1971).

Interpretation of the sex distribution of neoplasia in cattle tends to be complicated, in many countries, by the slaughter for human consumption of the vast majority of male animals at an early age. Nevertheless, it has been possible, by taking this factor into account, to identify a sexual bias in the frequency of a neoplasm as shown by Priester and Mantel (1971) who were able to demonstrate that the frequency of ocular squamous cell carcinoma is greater in females than in males.

The value of such observations is evident particularly in relation to the age distribution of lymphosarcoma and the breed distribution of ocular squamous cell carcinoma which were significant in the implication of a virus in the aetiology of certain forms of lymphosarcoma and the relationship between ocular squamous cell carcinoma and the lack of periorbital pigmentation.

The following section was undertaken to identify any factors of significance in the age, breed and sex distribution of the neoplasms under study which could contribute to the understanding of their aetiology.

MATERIALS AND METHODS

(1) Animals

The animals considered in this section are those

referred to in Chapter 1. The term 'immature' is applied to animals aged less than three years and 'adult' to animals aged three years and older.

(2) Statistical Methods

The statistical methods used were the correlation coefficient (Bishop, 1971) and the chi-squared test (Siegel, 1956). Unless otherwise stated, when a correlation or association is described as 'significant' this implies that the probability of its resulting from chance is less than two per cent ($p = <0.02$). When a correlation or association is described as "highly significant" this indicates that its probability of resulting from chance is less than 0.1 per cent ($p = <0.001$).

RESULTS

(1) Age Distribution

All admissions

An accurate age was available for 2420 of the 2809 animals admitted between 1/9/71 and 31/8/79. Ninety-three of the animals whose exact age was unknown were 'aged adults' or adults greater than seven years of age and the remaining 296 were 'adults' aged two years or older. In order to provide a basic age distribution of all

admissions against which prevalence rates could be calculated, the animals whose ages were not accurately known were allocated to age groups in the following manner. The 296 'adults' were distributed amongst the age groups between two years and 18 years in proportion to the numbers of animals of known age in these groups, and similarly, the 93 'aged adults' and adults aged greater than seven years were distributed amongst the age groups between eight and 18 years in proportion to the numbers of animals of known age in these groups. The resulting age distribution of all admissions is recorded in Figure 33.

Malignant neoplasms (All sites)

An accurate age was obtained for 226 of the 253 animals affected by malignant neoplasms and the age distribution of these animals is recorded in Figure 34. The 27 animals for which an accurate age was unavailable were all adults over four years old. Despite the large number of malignancies seen in immature animals the age specific prevalence rates indicate a pattern of comparatively low prevalence (<75 per 1000 admissions) in immatures and adults aged under eight years. In older animals, there is a rapid increase in the prevalence of malignancy with age and in those over 14 years the prevalence is in excess of 300 per 1000 admissions. A highly significant correlation ($p = <0.001$) exists between age and the prevalence of all malignancies.

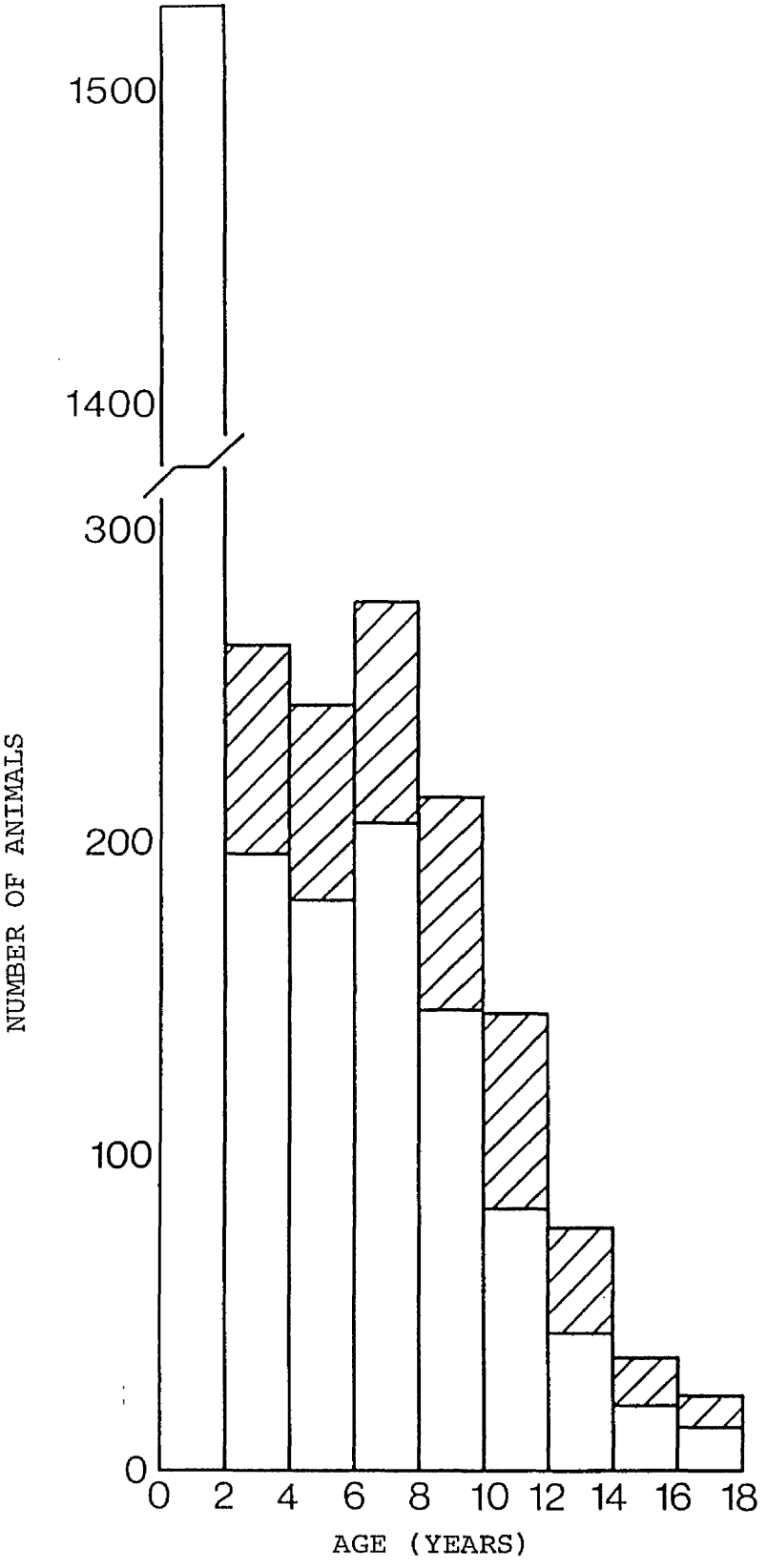


FIGURE 33 The age distribution of all admissions during the period 1/9/71 - 31/8/79. (The hatched areas indicate animals allocated to age groups as described in the text).

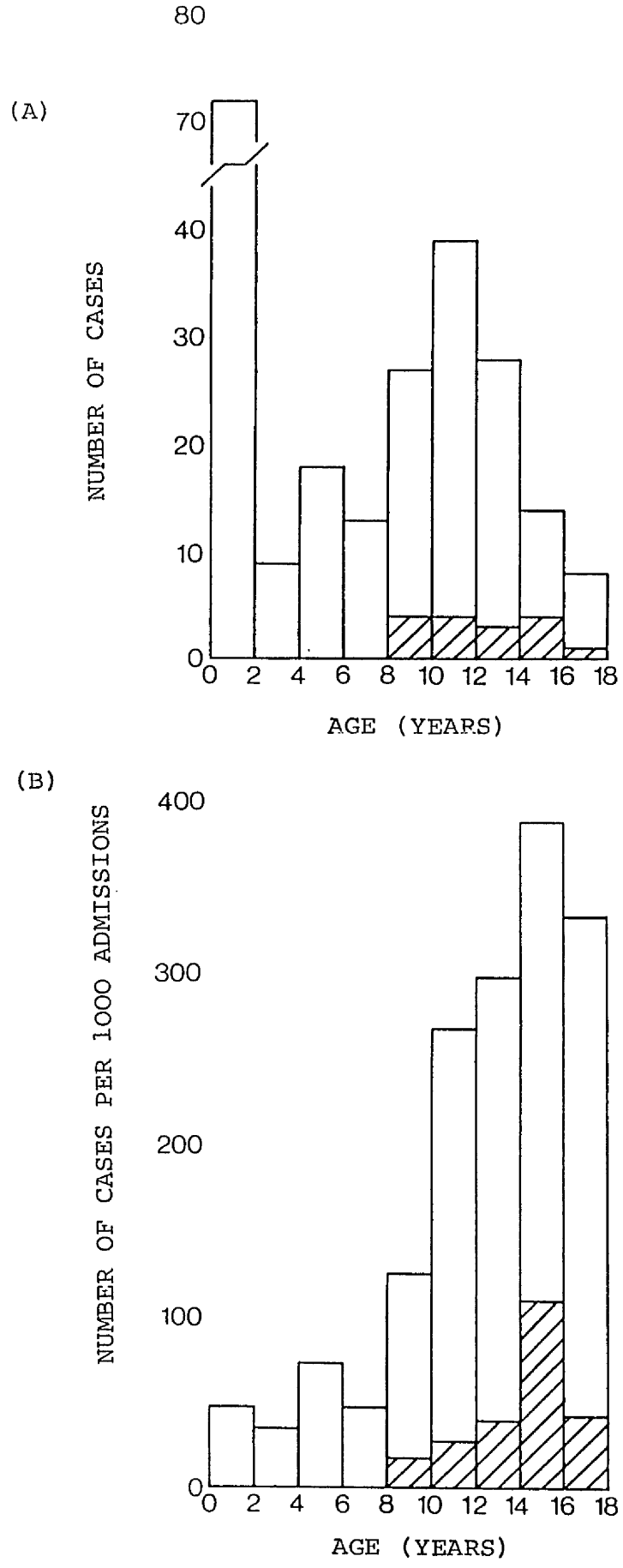


FIGURE 34 The age distribution (A) and age prevalence (B) of malignant neoplasms (all sites). (Hatched areas indicate animals with multiple malignancies

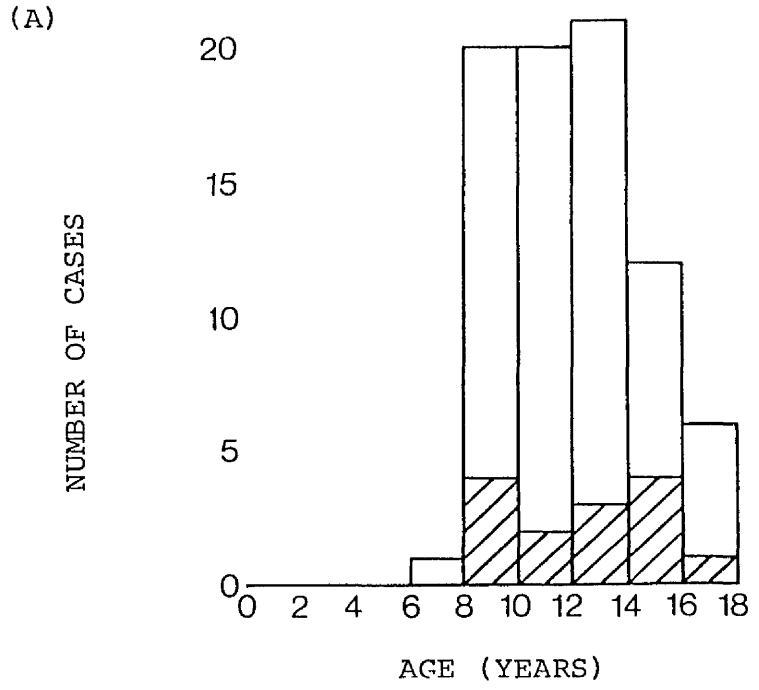
Multiple malignancies were only found in animals aged over seven years and the age distribution of these animals is recorded in Figure 34. There is an increasing prevalence of multiple malignancies with age (Figure 34) and a significant correlation ($p = <0.01$) exists between age and the prevalence of multiple malignancies.

Upper alimentary squamous cell carcinoma (UASCC)

An accurate age was obtained for 80 of the 97 animals affected by UASCC and all were adults aged over six years. The age distribution is recorded in Figure 35. A marked increase in the prevalence of UASCC with age is evident (Figure 35) and there is a highly significant correlation ($p = <0.001$) between age and the prevalence of UASCC. Sixty-six of the 80 animals of known age were affected by UASCC in the absence of any other malignancy and the prevalence of these cases increased with age (Figure 35). A highly significant correlation ($p = <0.001$) exists between age and the prevalence of UASCC in the absence of any other malignancy.

Upper alimentary papillomas (UAP)

An accurate age was obtained for 191 of the 234 animals affected by UAP and the age distribution of these animals is recorded in Figure 36. The youngest animal affected was aged 15 months and there was a steady increase in the prevalence of UAP from less than 20 per 1000 admissions in animals aged under four years to over 400 per 1000 admissions in animals aged in excess of 12 years



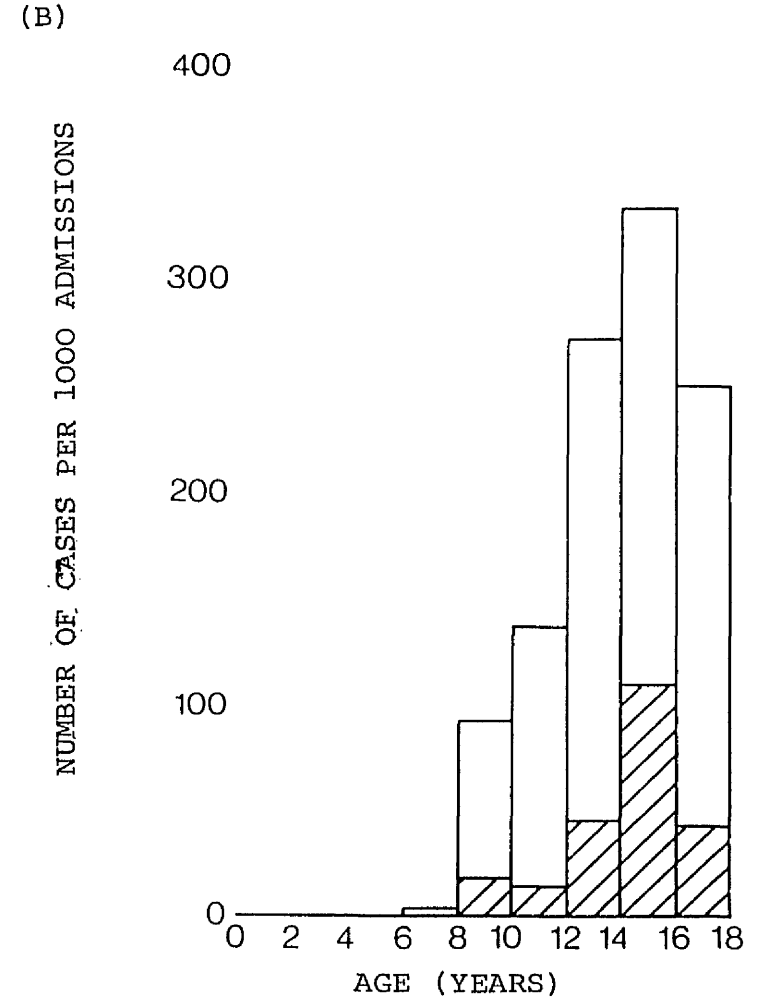


FIGURE 35 The age distribution (A) and age prevalence (B) of upper alimentary squamous cell carcinoma. (Hatched areas indicate other malignancy(ies) also present).

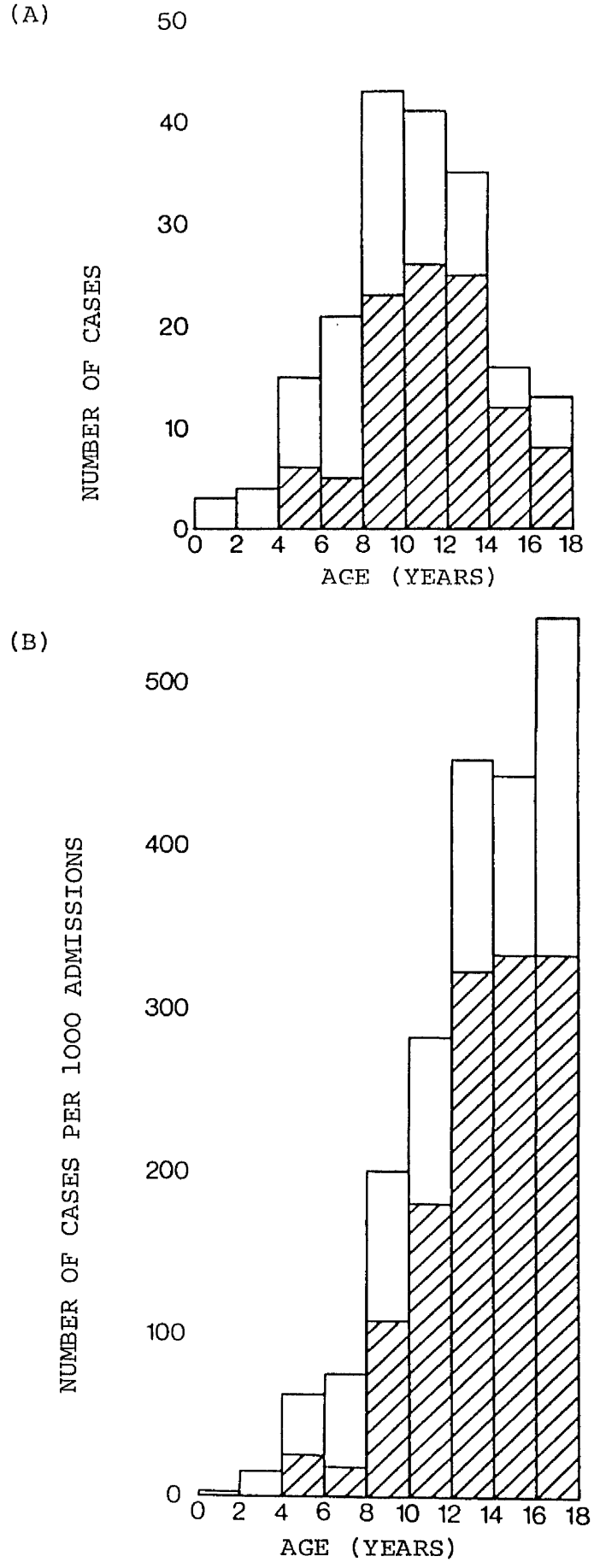


FIGURE 36 The age distribution (A) and age prevalence (B) of upper alimentary papillomas. (Hatched areas indicate malignancy(ies) also present).

(Figure 36). There is a highly significant correlation ($p = <0.001$) between age and the prevalence of UAP. No malignancy was present in 86 of the 191 animals of known age affected by upper alimentary papillomas. The prevalence of these cases (Figure 36) also increased with age and a highly significant correlation ($p = <0.001$) exists between age and the prevalence of UAP in the absence of malignancy.

Intestinal adenocarcinoma (IAC)

An accurate age was obtained for 16 of the 18 animals affected by IAC, and the age distribution of these animals is recorded in Figure 37. All the animals were aged between five and 15 years. The prevalence of IAC (Figure 37) tends to increase with age but there is no significant correlation between age and the prevalence of IAC.

Malignant urinary bladder neoplasms (MUBN)

An accurate age was obtained for 28 of the 31 animals affected by MUBN and the age distribution of these animals is recorded in Figure 38. The youngest animal was aged three years and the prevalence of MUBN (Figure 38) increases with age. A significant correlation ($p = <0.01$) exists between age and the prevalence of MUBN. However when only animals with MUBN in the absence of any other malignancy are considered, there is no correlation between age and the prevalence of MUBN.

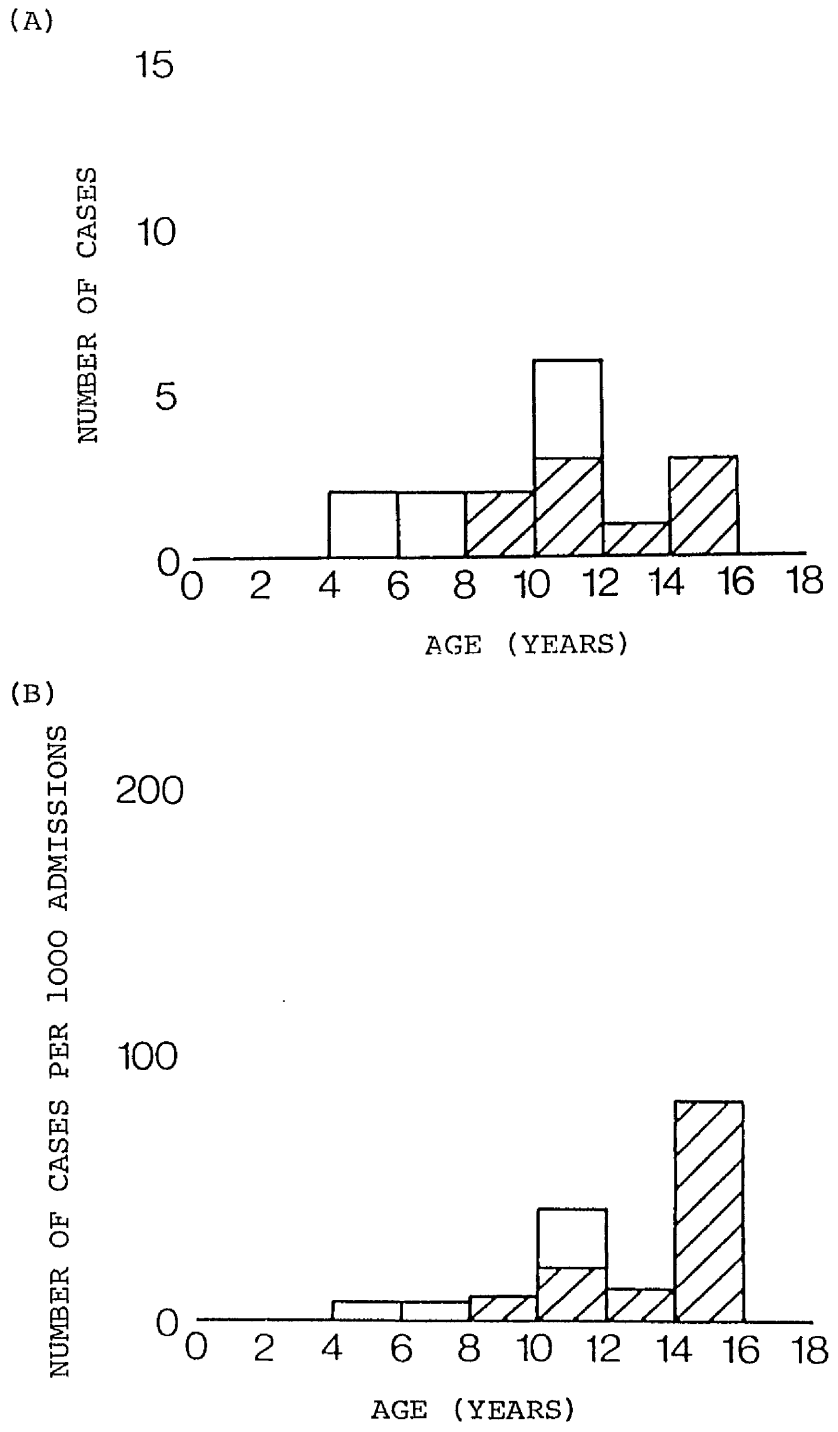


FIGURE 37 The age distribution (A) and age prevalence (B) of intestinal adenocarcinoma. (Hatched areas indicate other malignancy(ies) also present).

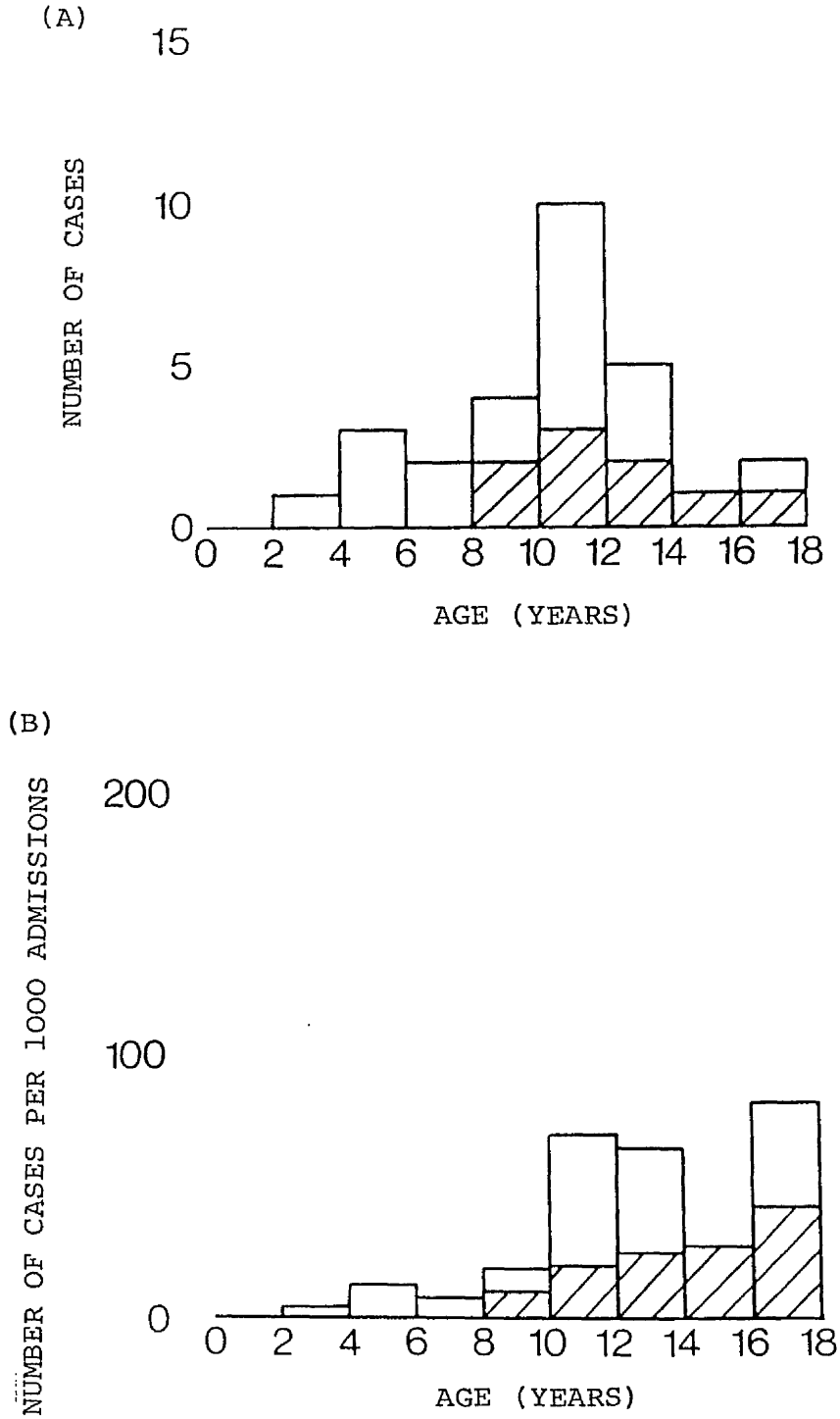


FIGURE 38 The age distribution (A) and age prevalence (B) of malignant urinary bladder neoplasia. (Hatched areas indicate other malignancy(ies) also present).

Benign urinary bladder neoplasms (BUBN)

An accurate age was obtained for 38 of the 41 animals affected by BUBN and the age distribution of these animals is recorded in Figure 39. The youngest animal was aged five years and the prevalence of BUBN (Figure 39) increases with age. A significant correlation ($p = <0.01$) exists between age and the prevalence of BUBN. However when only animals with BUBN in the absence of any malignancy are considered, there is no correlation between age and the prevalence of BUBN.

Lymphosarcoma

An accurate age was obtained for all 77 animals affected by lymphosarcoma and their age distribution is recorded in Figure 40. Sixty-nine of the animals were aged less than three years and the prevalence of lymphosarcoma (Figure 40) tends to decrease with age. There is a significant negative correlation ($p = <0.02$) between age and the prevalence of lymphosarcoma.

However, separate examination of the thymic and multicentric forms of lymphosarcoma indicates that there are substantial differences in their age distributions and prevalences. Thymic lymphosarcoma was confined to animals aged between four months and three years with 45 per cent less than one year of age and 45 per cent between one and two years of age. In contrast, multicentric lymphosarcoma was observed in animals aged between two weeks and 12 years with 61 percent less than one year of age and only 16 per cent between one and

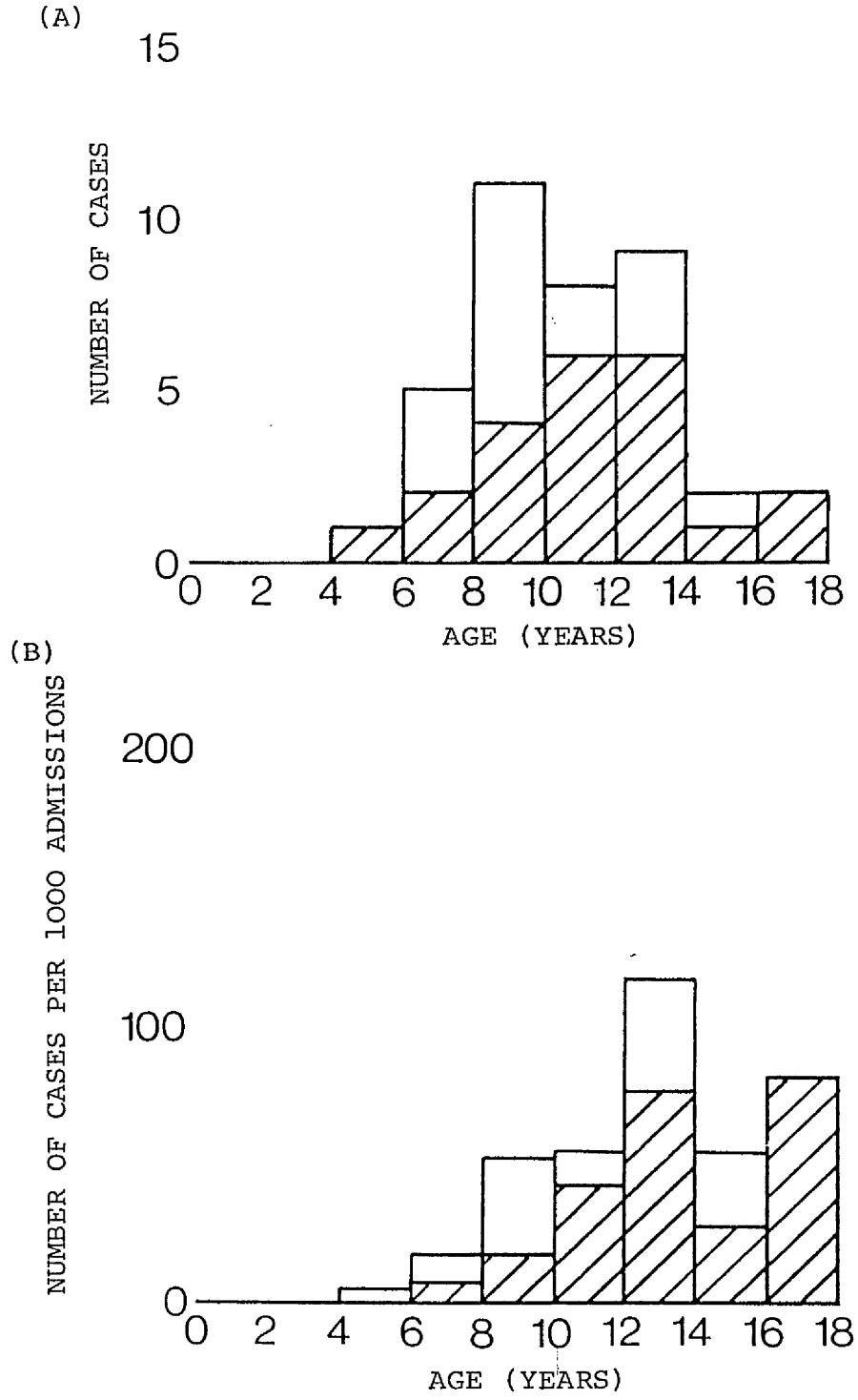


FIGURE 39 The age distribution (A) and age prevalence (B) of benign urinary bladder neoplasia. (Hatched areas indicate malignancy(ies) also present).

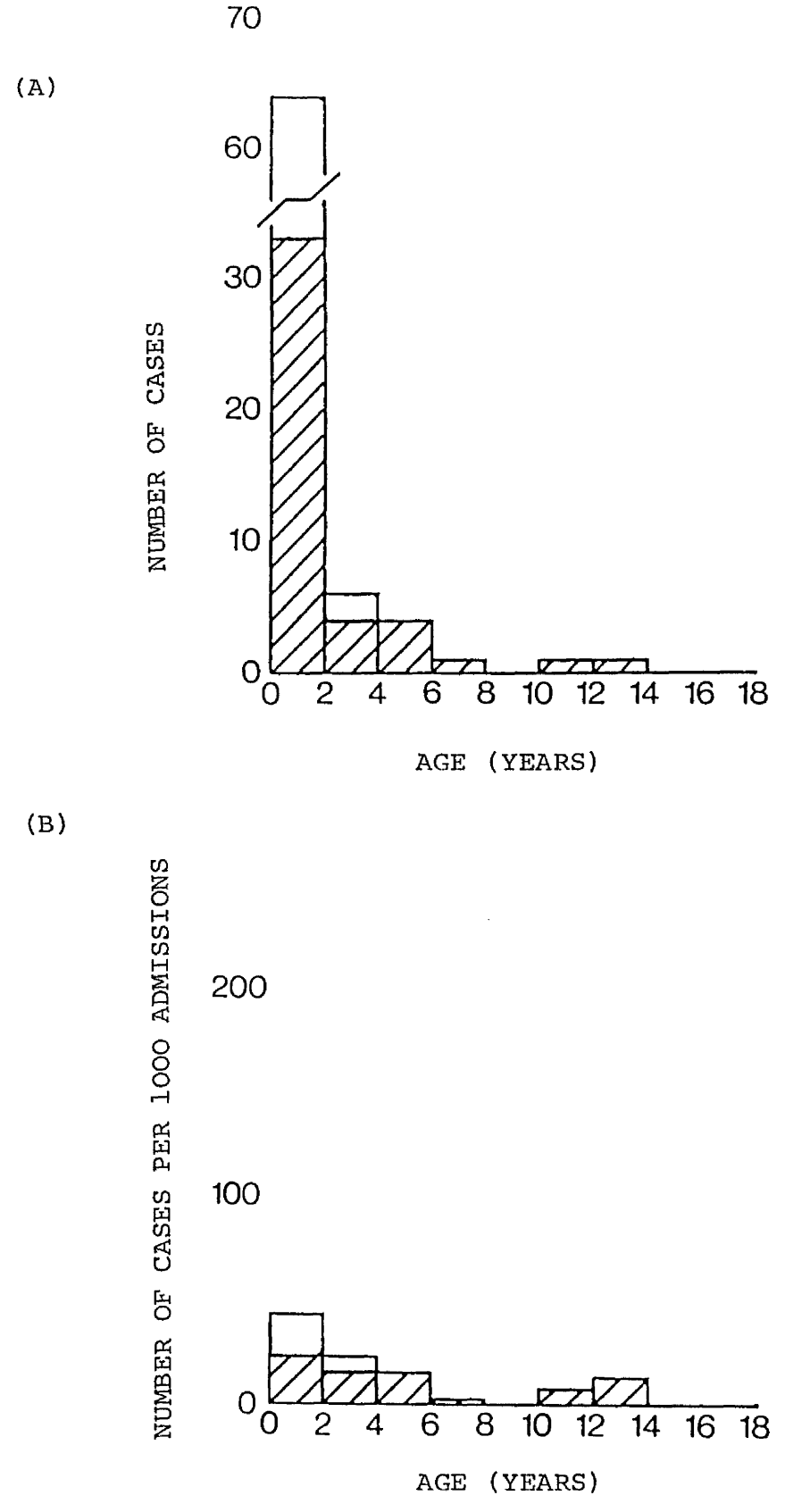


FIGURE 40

The age distribution (A) and age prevalence (B) of thymic lymphosarcoma (unhatched) and multicentric lymphosarcoma (hatched).

two years of age.

Other malignant neoplasms (OMN)

An accurate age was obtained for 44 of the 51 animals affected by other malignant neoplasms. Although there was a gradual increase in the prevalence of these neoplasms with age, the diverse nature of this group precludes interpretation of the age prevalence of any individual neoplasm.

(2) Type and Breed Distribution

The animals were categorised into either dairy or beef types according to their breeds. Dairy type animals accounted for 53.5 per cent of admissions and were represented by four breeds and their crosses; Friesian, Ayrshire, Jersey and Guernsey. Beef type animals accounted for 44.4% of admissions and the majority were represented by five breeds and their crosses; Hereford, Aberdeen Angus, Beef Shorthorn, Galloway and Highland. However due to the large numbers of cross bred animals of the latter four breeds it was frequently impossible to reasonably ascribe individual animals to any particular breed,and thus for the purposes of the following results these four breeds are considered as a single group. In addition, adult and immature animals are considered separately because the breed distributions within these age groups are dissimilar despite similar proportions of dairy and beef type animals. The type and breed of a small proportion (2.1%) of the animals admitted, all of which were immatures,was unknown or not recorded.

All adult admissions

Fifty-four per cent of all adult admissions were dairy type animals and 46 per cent were beef type animals. The Ayrshire and Friesian breeds each accounted for 46 per cent of the dairy type animals and the Jersey and Guernsey breeds for the remainder. Amongst the beef type animals, the majority (75%) were of the Aberdeen Angus, Beef Shorthorn, Galloway and Highland group of breeds with the

Hereford breed (22%) accounting for most of the remainder (Table 41).

Upper alimentary squamous cell carcinoma (UASCC)

Ninety-seven per cent of the animals affected by UASCC were of the beef type and the chi - squared test indicates a highly significant association ($p = < 0.001$) between this neoplasm and beef type animals. The breed distribution of UASCC also differed from that of all adult admissions in that the Hereford breed, which comprised over 20 per cent of the beef type animals admitted, accounted for only two per cent of those with UASCC (Table 41).

When only animals with UASCC in the absence of any other malignancy are considered the association between beef type animals and UASCC remains highly significant ($p = <0.001$) and the breed distribution is very similar to that found for all cases of UASCC.

Upper alimentary papillomas (UAP)

Eighty-eight per cent of the animals affected by UAP were of the beef type and the chi - squared test indicates a highly significant association ($p = <0.001$) between this neoplasm and beef type animals. As in animals affected by UASCC the proportion of beef type animals of the Hereford breed with UAP (10%) was less than would be expected when compared with the proportion of this breed in all adult admissions of the beef type (22%). The majority (71%) of the dairy type animals with UAP were of the

TABLE 41 Type and Breed Distribution of All Admission and Animals with Neoplasia

Case Group	Number of Animals	Dairy Types (%)	Beef Types (%)	Dairy Breeds			Beef Breeds		
				Friesian (%)	Ayrshire (%)	Jersey and Guernsey (%)	Hereford (%)	Shorthorn/Angus/ Highbland/Galloway (%)	Other Breeds (%)
All Admissions (Adult)	1277	54	46	46	46	8	22	75	3
OMN (Adult)	51	39	61	40	55	5	26	67	7
UASCC (All Cases)	97	3	97	0	100	0	2	98	0
UASCC (No Other malignancy)	80	2	98	0	100	0	3	97	0
UAP (All Cases)	234	12	88	29	71	0	10	89	1
UAP (No malignancy)	109	17	83	42	58	0	16	82	2
IAC (All Cases)	18	28	72	20	80	0	15	85	0
IAC (No other malignancy)	7	43	57	33	67	0	25	75	0
MUBN (All Cases)	31	23	77	14	86	0	8	92	0
MUBN (No other malignancy)	21	29	71	17	83	0	7	93	0
BUBN (All Cases)	41	15	85	17	83	0	11	85	3
BUBN (No malignancy)	17	18	82	33	67	0	14	79	7
Multiple Malignancy	19	10	90	0	100	0	6	94	0
All Admissions (Immature)	1532	53	43	65	34	1	53	34	13
OMN (Immature)	77	47	53	64	33	3	51	44	5

Ayrshire breed although this breed accounted for only 46 per cent of all adult admissions of the dairy type.

When only animals with UAP in the absence of any malignancy are considered, the association between beef type animals and UAP remains highly significant ($p = <0.001$). However the proportion of each breed in both the dairy and beef types approximated more closely to that for all adult admissions (Table 41).

Intestinal adenocarcinoma (IAC)

Seventy-two per cent of the animals affected by IAC were of the beef type and the chi- squared test indicates that there is some association ($p = < 0.05$) between this neoplasm and beef type animals. The breed distribution of IAC amongst the beef type animals did not vary markedly from the proportions in which the breeds were represented in all adult admissions (Table 41). Amongst the dairy type animals, the majority of those with IAC (80%) were of the Ayrshire breed but in view of the small numbers, any comparison with all adult admissions could be misleading.

When only animals with IAC in the absence of any other malignancy are considered no association is apparent between the neoplasm and beef type animals. However the number of animals in this category is inadequate for the chi^2 test to give reliable results.

Malignant urinary bladder neoplasms (MUBN)

Seventy-seven per cent of the animals affected by MUBN were of the beef type and the chi - squared test indicates a

highly significant association ($p = < 0.001$) between these neoplasms and beef type animals. The Hereford breed only accounted for eight per cent of beef type animals with MUBN as compared with 22 per cent of beef type animals admitted, and the majority (86%) of the dairy type animals with MUBN were of the Ayrshire breed although this breed accounted for only 46 per cent of all adult admissions of the dairy type.

When only animals with MUBN in the absence of any other malignancy are considered there is a significant association ($p = <0.02$) between beef type animals and MUBN and the breed distribution is very similar to that found for all cases of MUBN (Table 41).

Benign urinary bladder neoplasms (BUBN)

Eighty-five per cent of the animals affected by BUBN were of the beef type and the chi - squared test indicates a highly significant association ($p = <0.001$) between these neoplasms and beef type animals. The breed distribution amongst dairy and beef types was similar to that found in animals with MUBN (Table 41).

When only animals with BUBN in the absence of any malignancy are considered a significant association is maintained ($p = <0.01$) between beef type animals and BUBN.

Other malignant neoplasms in adults

Sixty-one per cent of the adult animals affected by OMN were of the beef type and 39 per cent were of the dairy type. The chi - squared test shows that there is some association ($p = < 0.05$) between these neoplasms and beef type animals. However the breed distribution amongst both beef and dairy types was similar to that for all adult admissions (Table 41).

All immature admissions

Dairy type animals accounted for 53 per cent of immature admissions and beef type animals for 43 per cent. The breeds represented were similar to those for adult animals but the proportions of the Friesian breed in the dairy type animals and the Hereford breed in the beef type animals were considerably greater (Table 41). The type and breed of a small proportion (4%) of the animals was unknown or not recorded.

Malignant neoplasms in immatures

Forty-seven per cent of the immature animals affected by malignancies were of the dairy type and 53 per cent were of the beef type. The chi - shared test shows that there is some association ($p = < 0.05$) between these neoplasms and beef type animals.However the distribution amongst the various breeds was very similar to that for all immature admissions (Table 41).

(3) Sex Distribution

In this study, all the adult animals affected by malignant neoplasia, upper alimentary papillomas and benign urinary bladder neoplasms were female. However, over 99 percent of adult admissions to the Medicine Department of the Veterinary Hospital during the period of the study were females. In addition, a high proportion (71%) of the immature animals with malignant neoplasms were female but the majority (63%) of all immature admissions were of this sex.

DISCUSSION

The patterns of age prevalence of the various neoplasms examined can be broadly divided into two categories, those in which there is an obvious increase in prevalence with age and those in which no increase in prevalence with age is apparent.

The prevalences of upper alimentary squamous cell carcinoma and upper alimentary papillomas steadily increase with age, and although upper alimentary papillomas can be observed in younger animals than upper alimentary squamous cell carcinoma, the overall shapes of the age prevalence curves are comparable. Similarly, the prevalences of malignant and benign urinary bladder neoplasms and intestinal adenocarcinoma increase with age but, probably due to the smaller numbers of cases, the slopes of the age prevalence curves are less well-defined.

A pattern of sustained increase in prevalence with age, which is particularly evident with regard to upper alimentary squamous cell carcinoma and upper alimentary papillomas, is similar to that observed in the age incidence of a wide range of cancers in man, including cancer of the oesophagus, small intestine and urinary bladder (Waterhouse, 1974). In addition,

it has been suggested that this pattern occurs when the major aetiological stimulus is an exogenous agent which acts continuously throughout life (Higginson and Muir, 1973).

In contrast, there is no apparent increase in the prevalence of either the thymic or multicentric forms of lymphosarcoma with age. The thymic form of lymphosarcoma was found exclusively in animals aged between four months and three years which is almost identical to the age distribution reported by Dungworth and others (1964). Similarly, Cotchin (1960), Jarrett and Crighton (1965) and Anderson, Jarrett and Crighton (1969) record that the majority of cases of thymic lymphosarcoma which they examined (93%, 86% and 73% respectively) were in animals under four years of age.

The multicentric form of lymphosarcoma was found in animals of all ages ranging between two weeks and 12 years, but the vast majority (84%) were less than four years of age. Cotchin (1960) and Jarrett and Crighton (1965) have reported a similar age distribution pattern for multicentric lymphosarcoma in the United Kingdom although the proportion under two years reported by the latter authors (45%) was less than found by Cotchin (1960) or in the present study (77% in both cases). In contrast, in countries where enzootic bovine leukosis has been recognised for many years, adult animals aged four years or greater account for between 80 and 90 percent of cases of multicentric lymphosarcoma (Marshak and others, 1962; Theilen, Dungworth, Lengyel and Rosenblatt, 1964; Bendixen, 1965). As in cattle, certain types of lymphoid neoplasms in man have a high prevalence in young age groups, for example, Burkitt's lymphoma (Burkitt, 1968) and acute lymphoblastic leukaemia (Zippin, Cutler, Reeves and

Lum, 1971) and it has been suggested that this pattern of age prevalence may indicate a possible viral aetiology with an increased prevalence in immunologically deficient children (Higginson and Muir, 1973). This hypothesis may also be applicable to multicentric and thymic lymphosarcoma in young cattle but at present there is no supporting epidemiological evidence, such as the clustering in space and time observed in Burkitt's lymphoma (Pike, Williams and Wright, 1976), for a viral aetiology in cattle (vide infra).

As with age prevalence there were a number of differences in the breed distribution of the various types of neoplasia examined. A higher than expected frequency of animals of beef breeds were affected by upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia. The most likely explanation for this bias towards beef breeds is that exposure to the aetiological stimuli responsible is related to the environment in which beef breeds of cattle are utilised. This would suggest that the aetiological stimuli are most prevalent in areas where the poorer qualities of pasture are found. The individual breed distribution also tends to support this premiss, particularly with respect to upper alimentary squamous cell carcinoma. A high proportion of cases of this neoplasm was found in breeds, such as the Highland and Galloway, and their crosses, which are specially suited to grazing poor quality marginal and hill pastures (Jardine, 1963; Aitchison, 1963) whereas a lower than expected frequency was found in the Hereford breed which is probably the breed of beef cattle most commonly utilised on improved pastures.

In the cases of intestinal adenocarcinoma, it is difficult

to draw any firm conclusions with regard to breed distribution as it has some similarity to those of the neoplasms already discussed when all cases are taken into account but, more closely resembles that of all adult admissions when only animals in which intestinal adenocarcinoma is the sole malignancy are considered.

The adult animals with other malignant neoplasms and the immature animals with malignant neoplasia both had a slightly higher frequency of beef types than expected, but the breed distributions within the dairy and beef types are very similar to those of all adult and all immature admissions respectively. The reason for the higher frequency of beef types in the adult animals is probably related to the fact that dairy cows tend to be culled at an earlier age than beef cows and thus fewer dairy cows live to an age when neoplasia is most prevalent. It would also seem likely that this factor contributes to the higher frequency of beef types in animals with upper alimentary squamous cell carcinoma, upper alimentary papillomas, urinary bladder neoplasia and intestinal adenocarcinoma.

With regard to the immature animals, in which lymphosarcoma accounted for over 90 percent of malignancies, it is evident that the higher frequency of beef types is due to the high proportion of beef animals (66%) with multicentric lymphosarcoma. There is no apparent explanation for this distribution which is in marked contrast to that found by Theilen and Dungworth (1965) who recorded eight dairy animals in their nine cases of multicentric lymphosarcoma in immatures. Similarly the results of this study, in which the thymic form of lymphosarcoma was found equally in dairy and beef types, contrast with the findings of Dungworth and others (1964) who record that 11 of their 14 cases were beef type animals, all but one of which were of the Hereford breed.

These authors could not account for the preponderance of Herefords amongst their cases and, in view of the results of the present study, it would seem probable that this unusual breed distribution was due to a factor such as a reflection of the breed distribution in the population at risk, rather than an increased susceptibility of the Hereford breed.

Although all adult animals and the majority of immature animals with malignant neoplasia were female there is no obvious increase in the frequency of females when the sex distribution of admissions is taken into account.

In summary, it has been shown that the neoplasms examined in detail exhibit various distinctive patterns of age prevalence and breed distribution. The patterns observed for upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia tend to suggest that the factor(s) involved in their aeitology are likely to act continuously throughout life and to be most prevalent in areas where beef cattle predominate. In light of these possibilities, the geographical distribution of bovine neoplasia is examined, in the following section, with particular reference to a known carcinogen, bracken fern.

SECTION II

A COMPARATIVE STUDY OF THE GEOGRAPHICAL DISTRIBUTION OF BOVINE NEOPLASIA AND BRACKEN FERN (*PTERIDIUM AQUILINUM*)

INTRODUCTION

The occurrence of urinary bladder neoplasia in cattle and the presence of bracken fern on the farms or in the areas of origin of affected animals has been reported by workers in various countries throughout the world. Similarly, a relationship between the occurrence of upper alimentary squamous cell carcinoma and the distribution of bracken fern has been described by workers in Brazil (Dobereiner and others, 1967; Campos Neto and others, 1975). However, these associations have invariably been proposed in the absence of statistical evaluation and, in the case of upper alimentary squamous cell carcinoma, the validity of an association has been challenged (Plowright and others, 1971).

The purpose of the following study is to examine whether there is any statistical relationship between the geographical distribution of the farms of origin of animals affected by neoplasms identified in the survey reported in Chapter 1 and the geographical distribution of bracken fern.

MATERIALS AND METHODS

(1) Selection of Animals

In this section of the study, only animals which

were referred from farms in Scotland were utilised. During the eight year study period a total of 2809 animals were admitted, of which 82.9 per cent were referred from farms in Scotland, the remainder being referred from farms in England (8.8%) or cattle dealers and markets (8.3%). The comparable figures for animals affected by malignant neoplasms were 92.1 per cent, 2.8 per cent and 5.1 per cent respectively (Table 42). Of the 254 animals with malignancies, 19 were affected by two or more different types and thus a total of 275 malignancies were identified. The numbers of the different types of malignancies which were found in animals from each of the three sources are recorded in Table 43,and those which were present in animals from Scottish farms (252) provide the basis for the results in this section. Similarly the sources of animals with upper alimentary papillomas and benign urinary bladder neoplasms are recorded in Table 44 and only those found in animals from Scottish farms are utilised in this section.

Details of the sources of individual animals are recorded in Appendix 4. (Case numbers E1 - E64, E66 - E148, E150 - E186, E188 - E203, E205 - E223, E225 - E245, E247 - E255 and E350 - E426).

TABLE 42

The Sources of All Bovine Admissions and of Animals with Malignant Neoplasia

		Scottish Farms	English Farms	Cattle Dealers or Markets	Totals
All Bovine Admissions	Adults	978	170	129	1277
	Immatures	1350	77	105	1532
Animals with Malignant Neoplasia	Adults	158	6	13	177
	Immatures	76	1	0	77

TABLE 43

The Sources of Individual Types of Malignant Neoplasms

	Scottish Farms	English Farms	Cattle Dealers or Markets	Totals
Upper Alimentary Squamous Cell Carcinoma	83	1	13	97
Malignant Urinary Bladder Neoplasms				
(i) Transitional Cell Carcinoma	22	2	0	24
(ii) Haemangiosarcoma	3	1	1	5
(iii) Adenocarcinoma	2	0	0	2
(iv) Squamous Cell Carcinoma	1	0	0	1
Intestinal Adenocarcinoma	15	1	2	18
Other Malignant Neoplasms including Lymphosarcoma (adults)	49	2	0	51
Other Malignant Neoplasms including Lymphosarcoma (immatures)	76	1	0	77
TOTAL (Adults)	176	7	15	198
TOTAL (Immatures)	76	1	0	77

TABLE 44

The Sources of Animals with Upper Alimentary Papillomas and Benign Urinary Bladder Neoplasms

	Scottish Farms	English Farms	Cattle Dealers or Markets	Totals
Upper Alimentary Papillomas	194(109)*	10(3)	30(13)	234(125)
Benign Urinary Bladder Neoplasms				
(i) Haemangiomas	36(21)	2(1)	0(0)	38(22)
(ii) Fibromas	6(3)	1(0)	0(0)	7(3)

* Figures in brackets are the number of animals which also had malignancies.

(2) Geographical Division of Scotland

In order to simplify descriptions of geographical locations in this section, Scotland has been divided into five regions as shown in Figure 41. The counties from which the regions are composed and the proportion of the total bracken infestation of the country (approximately 450,000 acres) in each region based on data recorded by Hendry (1958) is as follows :

(1) Northern region - Caithness, Inverness, Orkney, Ross and Cromarty, Shetland, Sutherland - 14.1 per cent,

(2) Western region - Argyll, Bute, Dunbarton, Perth - 43.9 per cent,

(3) Eastern region - Aberdeen, Angus, Banff, Moray, Nairn - 3.8 per cent,

(4) Central region - Ayr, Berwick, Clackmannan, East Lothian, Fife, Kincardine, Kinross, Lanark, Mid Lothian, Renfrew, Stirling, West Lothian - 9.3 per cent,

(5) Southern region - Dumfries, Kirkcudbright, Peebles, Roxburgh, Selkirk, Wigton - 28.9 per cent.

(3) Geographical Distribution of Bracken

The geographical distribution of areas of severe, moderate and light or nill bracken infestation throughout Scotland is recorded in Figure 42 which is an adaptation of the data recorded by Hendry (1958).

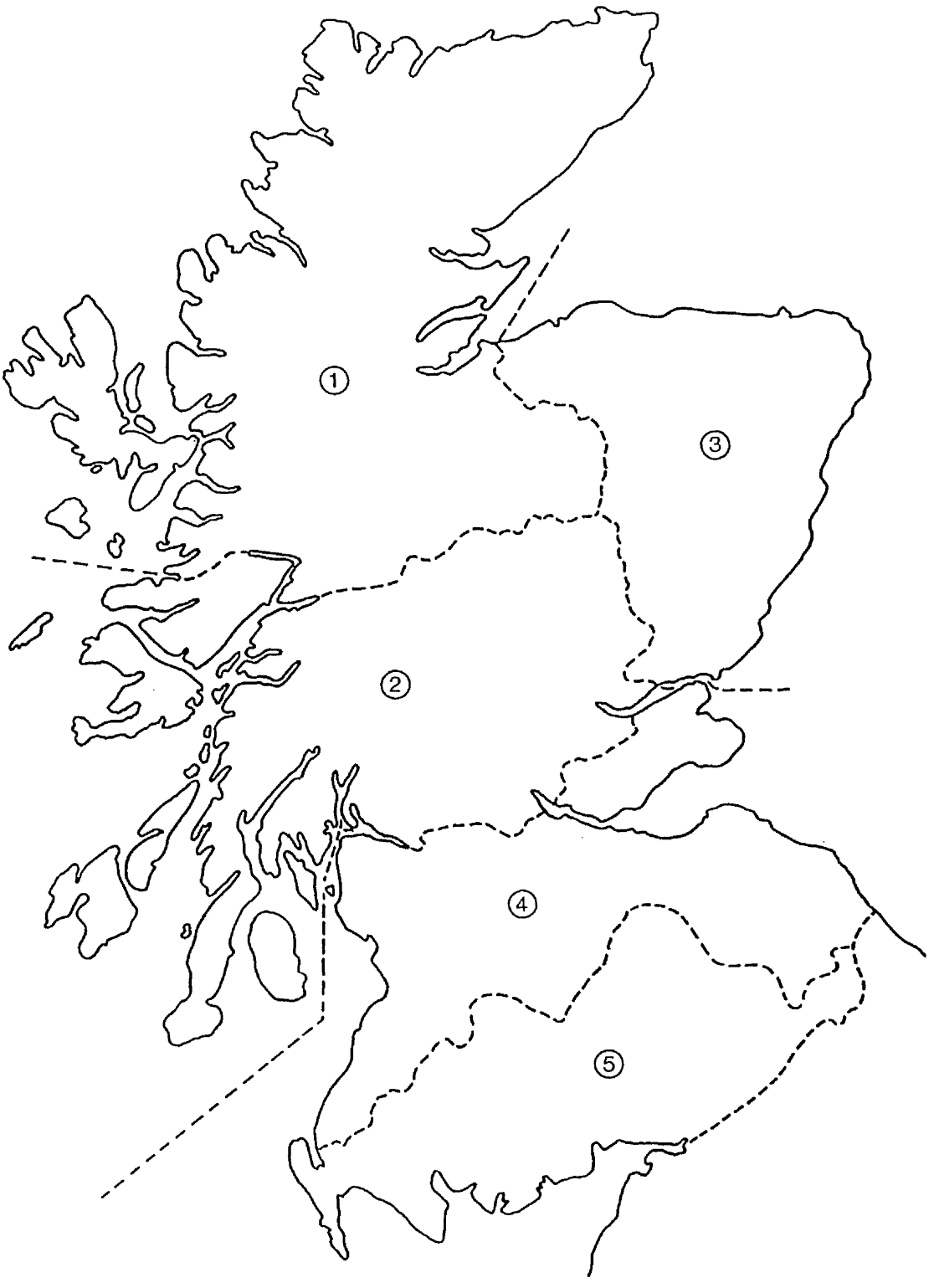


FIGURE 41 A Regional Division of Scotland

1. Northern Region
2. Western Region
3. Eastern Region
4. Central Region
5. Southern Region

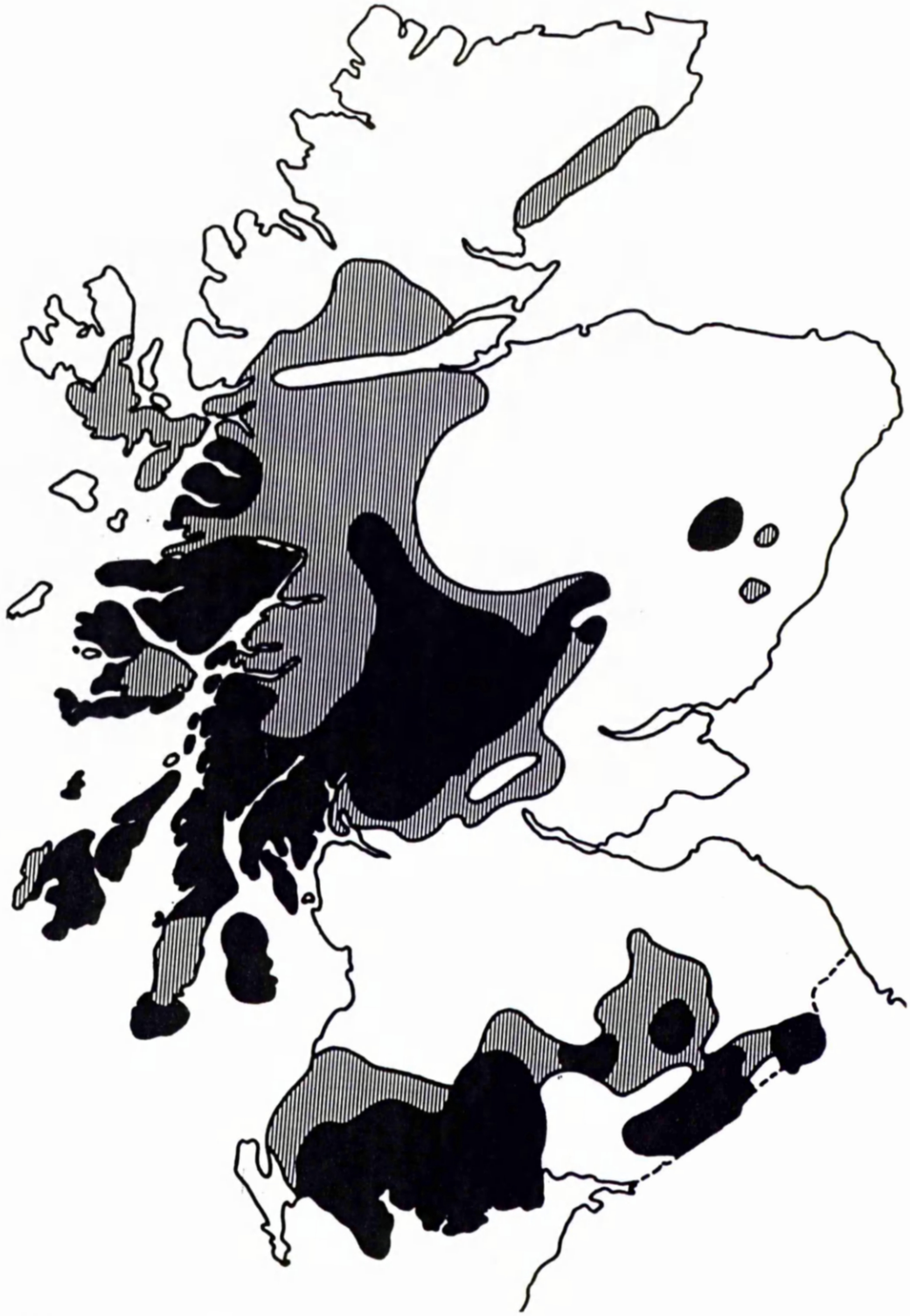


FIGURE 42 The Geographical Distribution of Bracken Fern (Pteridium agilinum) in Scotland (Adapted from G.W. Hendry, 1958).

Nil or light infestation

Moderate infestation

Severe infestation

(4) Statistical methods

The statistical method used was the chi-squared test. The data were tabulated into frequency tables and the chi-squared test performed as described by Siegel (1956). Unless otherwise stated, when an association is described as "significant" this implies that the probability of its resulting from chance is less than two per cent ($p = <0.02$). When an association is described as "highly significant" this indicates that its probability of resulting from chance is less than 0.1 per cent ($p = <0.001$).

RESULTS

All admissions of adult cattle

The geographical distribution of the referral farms of the 978 adult cattle admitted from farms in Scotland between 1.9.71 and 31.8.79 is recorded in Figure 43 and Table 45. 93.1 per cent originated in three regions: the central region (55.2%), the western region (24.7%) and the southern region (13.2%). When the geographical distribution of the referral farms of all adult admissions (Figure 43) is compared with the geographical distribution of bracken fern (Figure 42) it is found that 61 per cent are situated in areas of light or nil bracken infestation, 10 per cent in areas of moderate infestation and 29 per cent in areas of severe infestation.

Upper alimentary squamous cell carcinoma (UASCC)

The geographical distribution of the referral farms of 83 animals affected by UASCC is recorded in Figure 44 and Table 45. 81.9 per cent of the animals originated

TABLE 45

The Regional Distribution of the Referral Farms of Cattle Admitted during the Period 1.9.71 to 31.8.79

Case Groups	Number of Animals	Percentage of Animals from Individual Regions				
		Northern	Western	Eastern	Central	Southern
All Admissions (Adult)	978	4	25	3	55	13
UASCC (All Cases)	83	10	82	1	5	2
UASCC (No Other Malignancy)	69	6	84	1	6	3
UAP (All Cases)	194	13	72	1	8	6
UAP (No Malignancy)	85	14	65	1	13	7
IAC (All Cases)	15	13	67	0	7	13
IAC (No Other Malignancy)	6	0	50	0	17	33
MUBN (All Cases)	27	30	52	0	4	15
MUBN (No Other Malignancy)	19	32	42	0	5	21
BUBN (All Cases)	39	26	56	0	5	13
BUBN (No Malignancy)	17	24	59	0	6	12
OMN (All Cases)	49	6	41	2	41	10
OMN (No UAP)	36	6	33	3	53	6
All Admissions (Immature)	1350	2	8	6	71	13
All Malignancies (Immature)	76	3	8	8	60	21

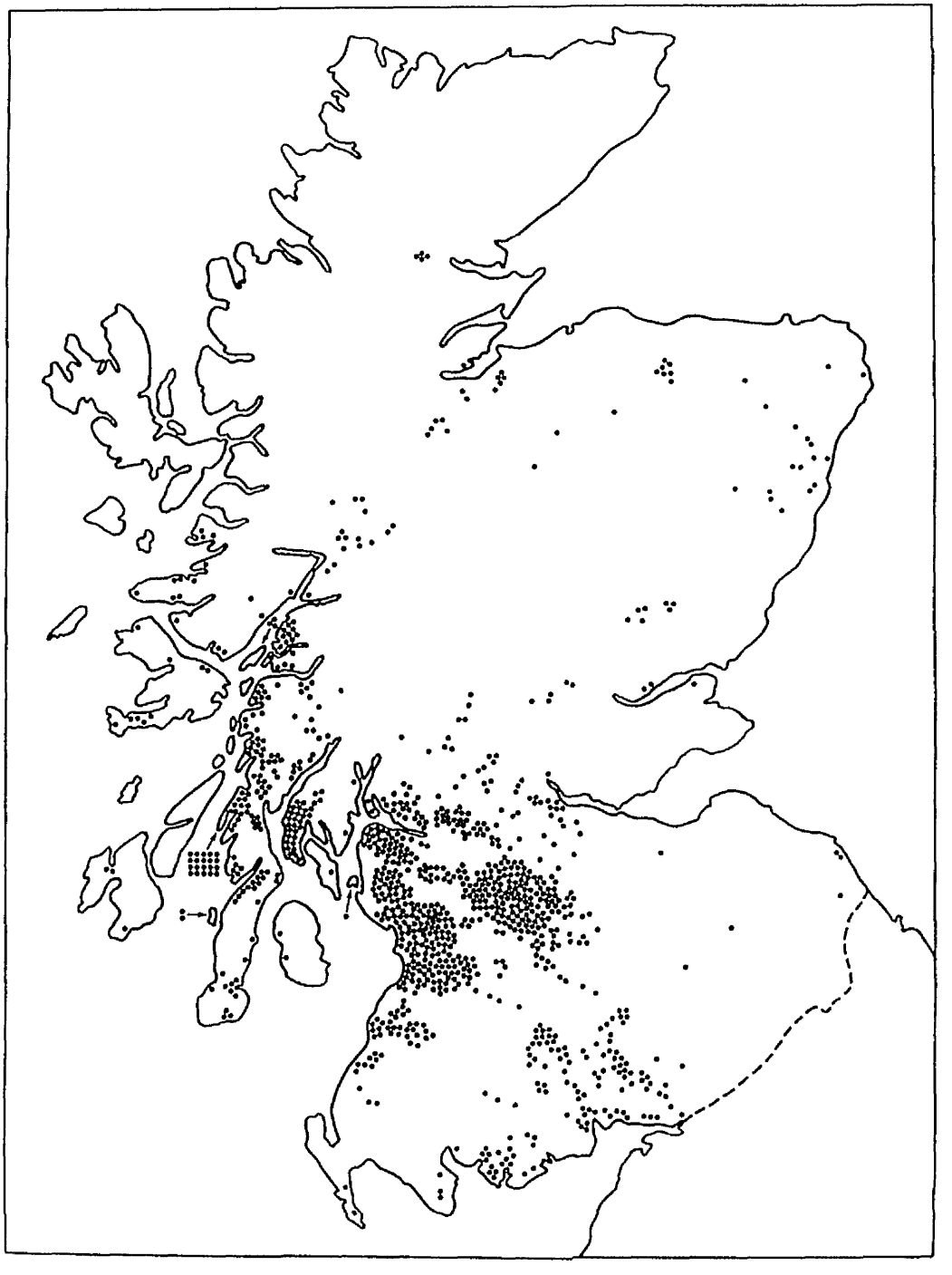


FIGURE 43 The Geographical Distribution of the Referral Farms of 978 Adult Cattle Admitted during the Period 1.9.71 to 31.8.79.

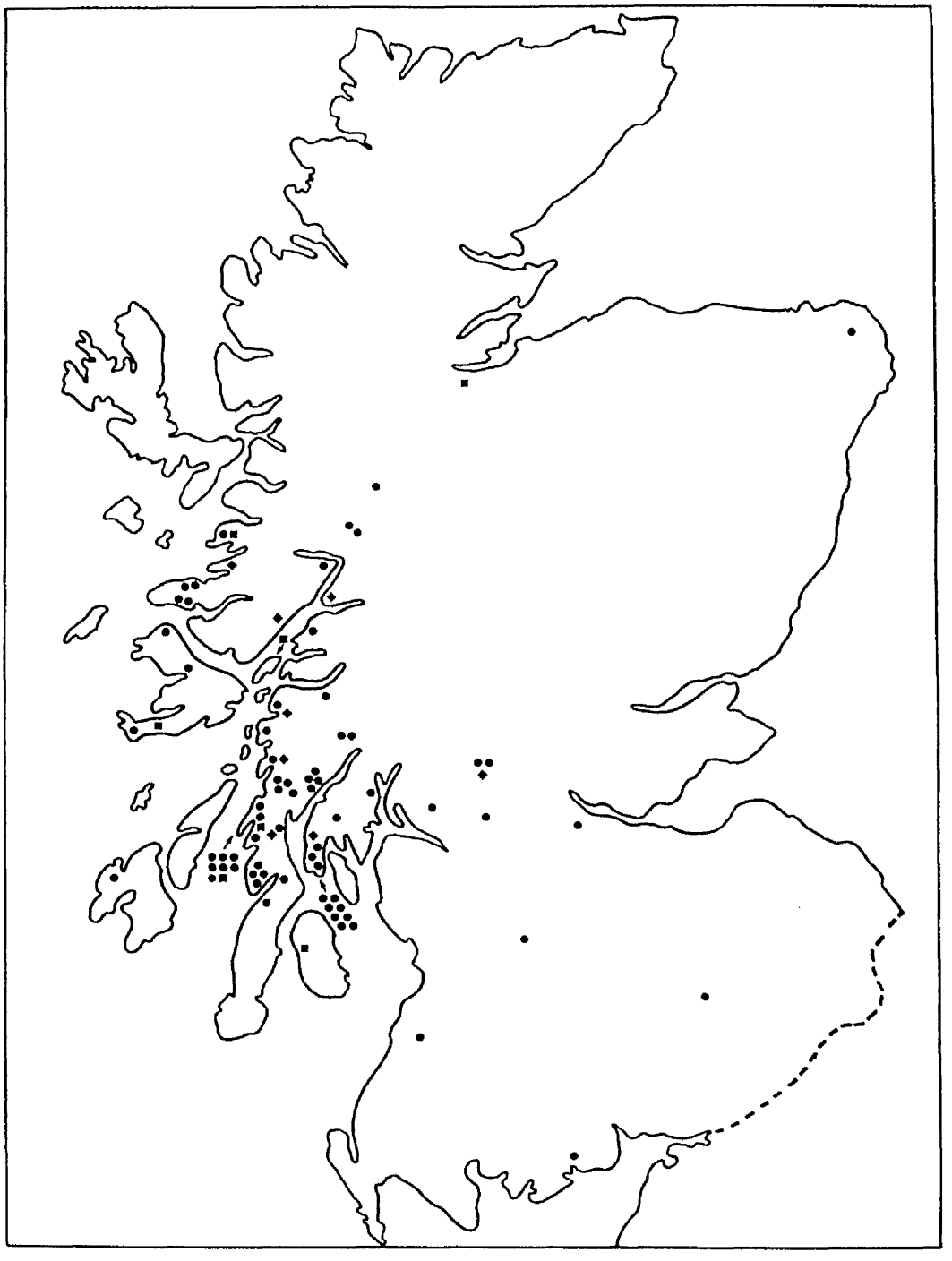


FIGURE 44 The geographical distribution of the referral farms of 83 cattle with upper alimentary squamous cell carcinoma. (Epidemiology case numbers: E1-E64 and E66-E84, Appendix 4).

- ● Upper alimentary squamous cell carcinoma
- ◆ Upper alimentary squamous cell carcinoma and intestinal adenocarcinoma
- ■ Upper alimentary squamous cell carcinoma and malignant urinary bladder neoplasia

in the western region, although this region accounted for only 24.7 per cent of adult admissions. The majority of the referral farms of the animals with UASCC from this region were concentrated along the western coast but this area accounted for the vast majority of the admissions from the region. 9.3 per cent of the animals with UASCC were referred from the northern region but most of these cases originated in the south western area of this region adjacent to the high concentration of cases in the western region. Although the central region accounted for 55.2 per cent of all adult admissions, only 4.8 per cent of the animals with UASCC originated in this region. When only animals affected by UASCC in the absence of any other malignancy are considered the proportion of cases from each region is very similar to that for all animals with UASCC (Table 45).

Comparison of the geographical distribution of the referral farms of animals affected by UASCC (Figure 44) with the geographical distribution of bracken fern (Figure 42) reveals that, in marked contrast with all adult admissions, 85 per cent of the animals with UASCC were referred from areas of severe bracken infestation and only six per cent from areas of light or nil infestation. The chi-squared test (Table 46) indicates that there is a highly significant association ($p = <0.001$) between this carcinoma and the severity of bracken infestation in the referral area. Similarly, when only animals with UASCC in the absence of any other malignancy are considered, the association remains highly significant (Table 46).

TABLE 46

The Severity of Bracken Infestation in the Areas of the Referral Farms of Animals with Upper Alimentary Squamous Cell Carcinoma Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
UASCC (All cases)	70	8	5	<0.001
UASCC (No other malignancy)	59	5	5	<0.001
All Adult Admissions	287	97	594	-

Upper alimentary papillomas (UAP)

The geographical distribution of the referral farms of 194 animals affected by UAP is recorded in Figure 45 and Table 45 and is similar to that of animals affected by UASCC. The majority (71.6%) of the animals with UAP originated in the western region and in particular, the western half of the region. 12.9 per cent were referred from the northern region but, as with animals affected by UASCC, the proportion referred from the central region was small (8.2%) considering the large number of all adult admissions which originated in this region. When only animals with UAP in the absence of any malignancy are considered the proportion of cases from each region is similar to that for all animals with UAP (Table 45), although there was a slight reduction, to 64.7 per cent, in the proportion from the western region and a slight rise, to 12.9 per cent, in the proportion from the central region.

When the geographical distribution of the referral farms of animals affected by UAP (Figure 45) is compared with the geographical distribution of bracken fern (Figure 42) it is found that 71 per cent of the animals with UAP were referred from areas of severe bracken infestation and only 12 per cent from areas of light or nil infestation. The chi-squared test (Table 47) indicates that there is a highly significant association ($p = <0.001$) between these neoplasms and the severity of bracken infestation in the referral area. When only animals with UAP in the absence of any malignancy are considered the proportion of animals

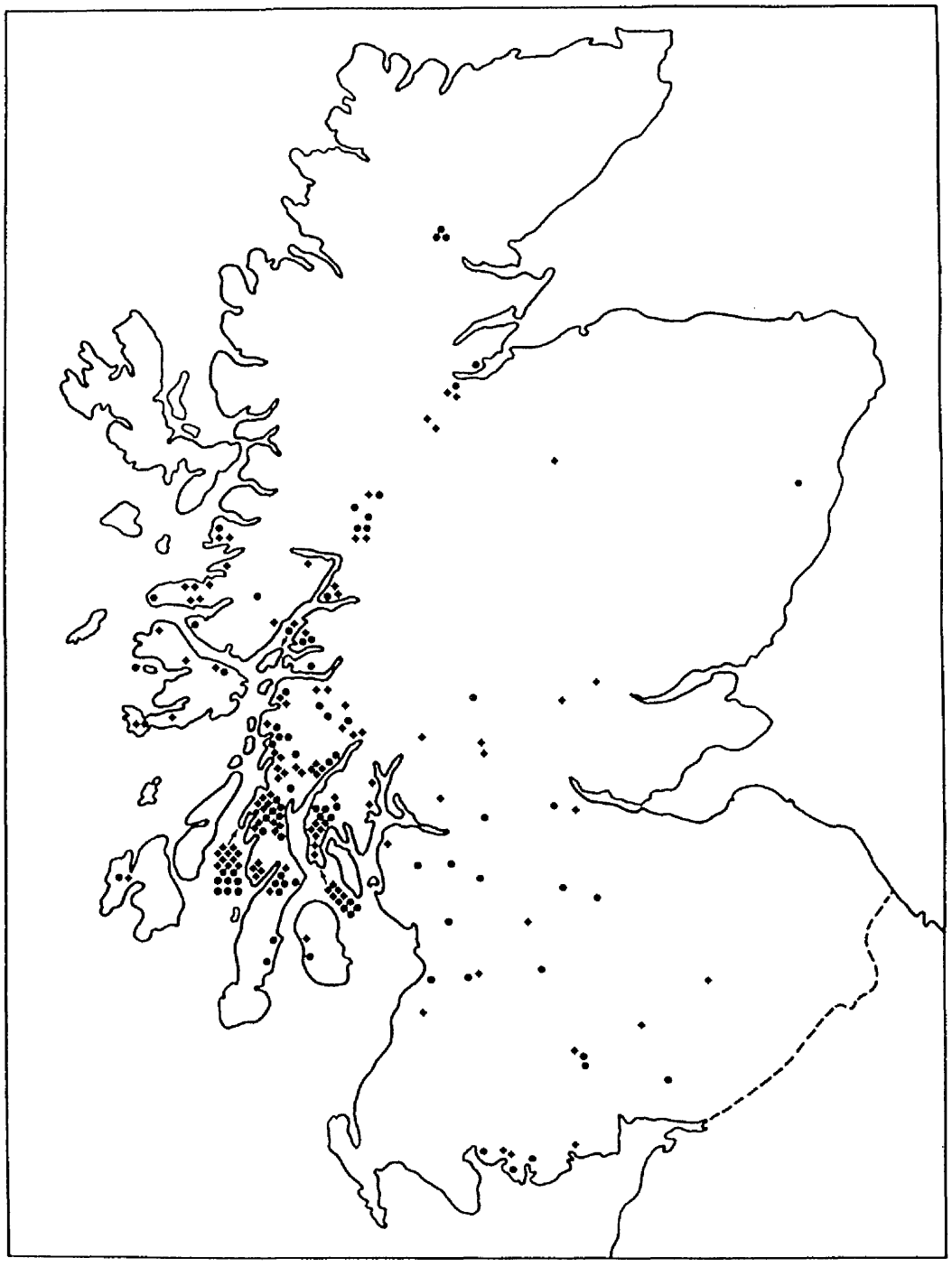


FIGURE 45 The Geographical Distribution of the Referral Farms of 194 Cattle and Upper Alimentary Papillomas (Epidemiology Case Numbers: E1-E8, E10-E58, E61-E64, E66-E147, E150-E160, E162, E165, E168-E170, E172, E174, E176-E180, E182-E186, E188-E191, E193-E195, E197-E200, E203, E212, E215, E218, E220, E221, E225, E228, E232, E236, E238 and E254, Appendix 4).

- ● Upper alimentary papillomas in the absence of malignancy.
- ◆ Upper alimentary papillomas in the presence of malignancy(ies).

TABLE 47

The Severity of Bracken Infestation in the Areas of the Referral Farms of Animals with Upper Alimentary Papillomas Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
UAP (All Cases)	138	33	23	<0.001
UAP (No Malignancy)	54	17	14	<0.001
All Adult Admissions	287	97	594	-

from areas of severe infestation is slightly reduced to 64 per cent but a highly significant association ($p = <0.001$) still exists between upper alimentary papillomas and the severity of bracken infestation in the referral area (Table 47).

Intestinal adenocarcinoma (IAC)

The geographical distribution of the referral farms of 15 animals affected by IAC is recorded in Figure 46 and Table 45. The majority (66.7%) originated in the western region and as with animals affected by UASCC and UAP only a small proportion (6.7%) was referred from farms in the central region. There were only six animals in which IAC was not accompanied by any other malignancy and their origins were widely scattered, three being in the western region, two in the southern region and one in the central region.

When the geographical distribution of the referral farms of animals affected by IAC (Figure 46) is compared with the geographical distribution of bracken fern (Figure 42) it is found that 60 per cent of the animals affected by intestinal adenocarcinoma were referred from areas of severe bracken infestation, 33 per cent from areas of moderate infestation and the remainder from areas of light infestation. The chi squared test (Table 48) indicates that there is a highly significant association ($p = <0.001$) between this neoplasm and the severity of bracken infestation in the referral area. However, 10 of the 15 examples of IAC were present in animals which were also affected by

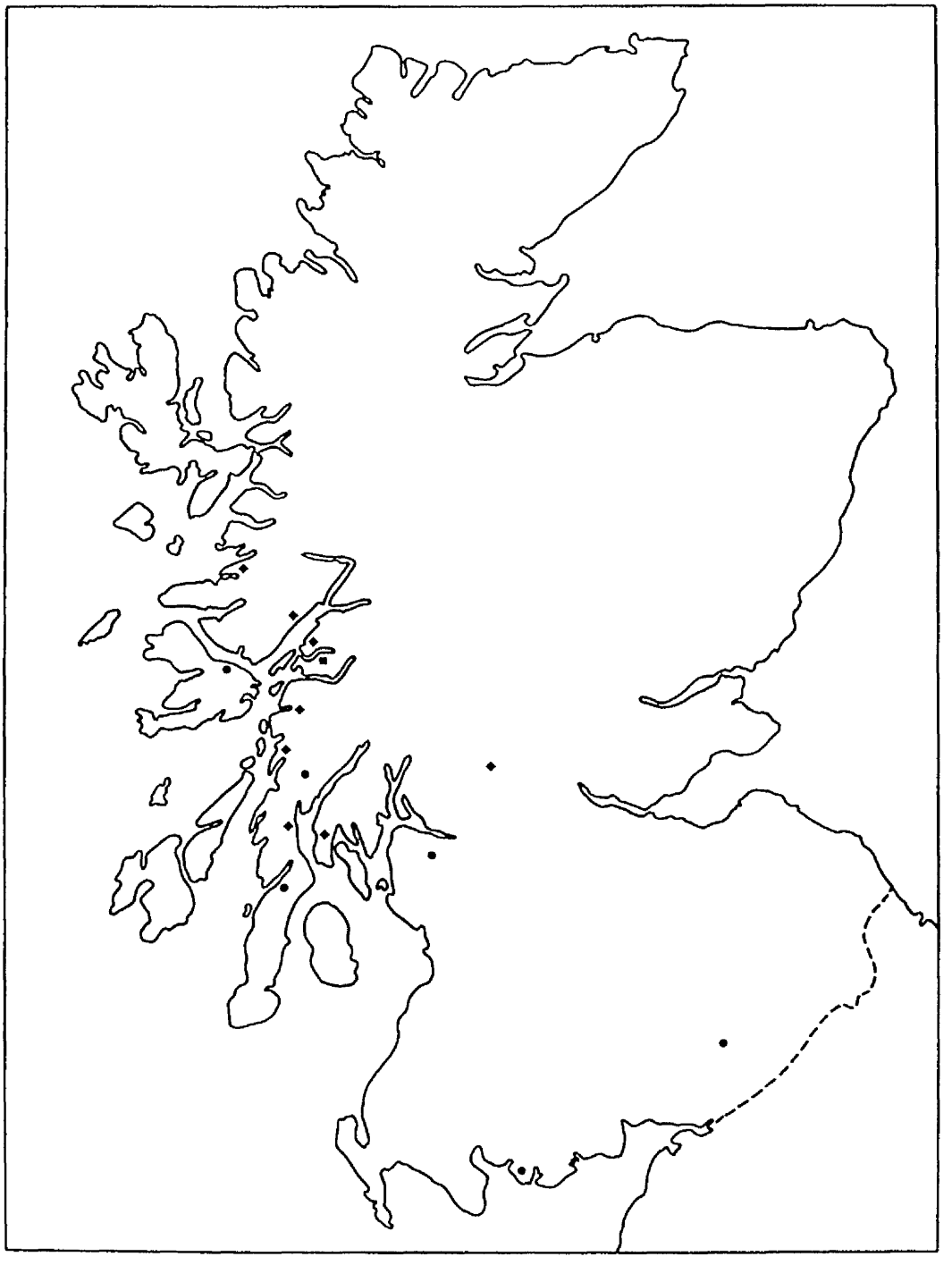


FIGURE 46 The Geographical Distribution of the Referral Farms of 15 cattle with Intestinal Adenocarcinoma.

(Epidemiology case numbers: E13, E15, E35, E42, E58, E62, E76, E82, E161-E165, E171 and E195, Appendix 4).

- ● Intestinal adenocarcinoma
- ◆ Intestinal adenocarcinoma and upper alimentary squamous cell carcinoma
- ■ Intestinal adenocarcinoma and malignant urinary bladder neoplasia

TABLE 48

The Severity of Bracken Infestation in the Areas of the Referral Farms of Animals with Intestinal Adenocarcinoma Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
IAC (All Cases)	9	5	1	<0.001
IAC (No Other Malignancy)	4	0	1	*
All Adult Admissions	287	97	594	-

* Chi-squared test unreliable. Three cells with expected frequencies less than five.

other malignancies, particularly UASCC. Consequently it was not possible to determine whether there is any association between IAC in the absence of any other malignancy and the severity of bracken infestation in the referral area.

Malignant urinary bladder neoplasms (MUBN)

The geographical distribution of the referral farms of 27 animals affected by MUBN is recorded in Figure 47 and Table 45. The majority (81.5%) originated in the western and northern regions from which 51.9 per cent and 29.6 per cent of the cases were referred respectively. Four cases (14.8%) originated in the southern region but only one (3.7%) in the central region. When only animals affected by MUBN but without any other malignancy are considered the proportion of cases from each area is similar to that for all animals with MUBN, although there was a slight reduction, to 41.1 per cent, in the proportion from the western region and a slight rise, to 21 per cent, in the proportion from the southern region.

Comparison of the geographical distribution of the referral farms of animals affected by MUBN (Figure 47) and the geographical distribution of bracken fern (Figure 42) reveals that 70 per cent of the animals affected by malignant urinary bladder neoplasms were referred from areas of severe bracken infestation, 19 per cent from areas of moderate infestation and 11 per cent from areas of light or nil infestation. The chi-squared test (Table 49) indicates

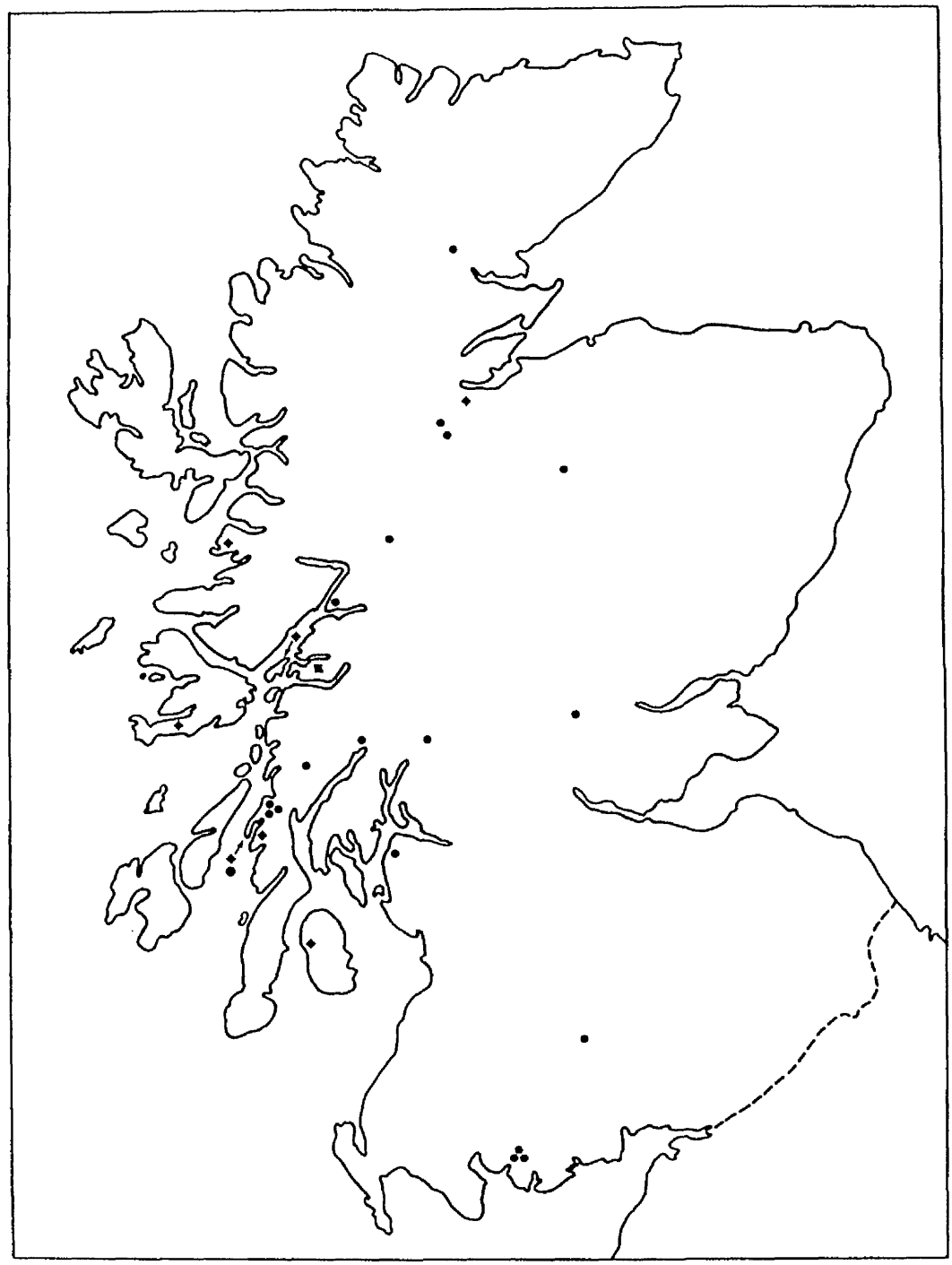


FIGURE 47 The Geographical Distribution of the Referral Farms of 27 Cattle and Malignant Urinary Bladder Neoplasia.

(Epidemiology case numbers: E69, E72, E73, E75, E77, E78, E80, and E166-E185, Appendix 4).

- ● Malignant urinary bladder neoplasia
- ◆ Malignant urinary bladder neoplasia and upper alimentary squamous cell carcinoma
- ■ Malignant urinary bladder neoplasia and intestinal adenocarcinoma

TABLE 49

The Severity of Bracken Infestation of the Areas of the Referral Farms of Animals with Malignant Urinary Bladder Neoplasia Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
MUBN (All Cases)	19	5	3	<0.001
MUBN (No Other Malignancy)	11	5	3	<0.001
All Adult Admissions	287	97	594	-

that there is a highly significant association ($p = <0.001$) between these neoplasms and the severity of bracken infestation in the referral area. When only animals with malignant urinary bladder neoplasia in the absence of any other malignancy are considered the proportion of cases from areas of severe bracken infestation is reduced to 58 per cent but the proportion from areas of moderate infestation increases to 26 per cent. However, a highly significant association ($p = <0.001$) still exists between malignant urinary bladder neoplasms and the severity of bracken infestation in the referral area (Table 49).

Benign urinary bladder neoplasms (BUBN)

The geographical distribution of the referral farms of 39 animals affected by BUBN is recorded in Figure 48 and Table 45. The proportion of cases referred from each region was very similar to that for animals affected by MUBN with 56.4 and 25.6 per cent originating from the western and northern regions respectively, and most of the remainder (12.9%) from the southern region. An almost identical pattern is apparent when only animals with BUBN but without any malignancy are considered (Table 45).

Comparison of the geographical distribution of the referral farms of animals affected by BUBN (Figure 48) and the geographical distribution of bracken fern (Figure 42) reveals that 62 per cent of the animals affected by benign urinary bladder neoplasms were referred from areas of severe bracken infestation, 28 per cent from areas of moderate infestation and 10 per cent from areas of light or

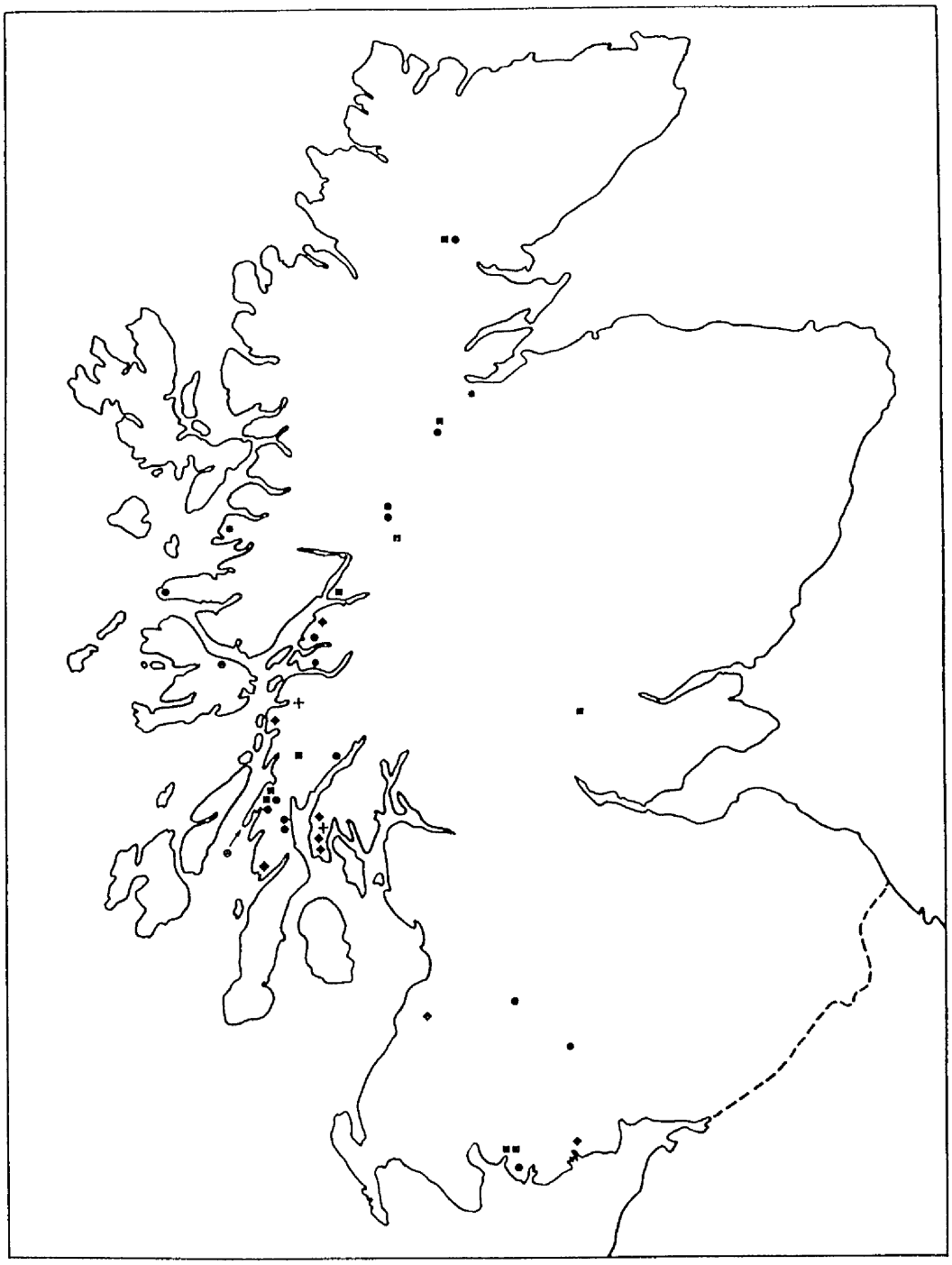


FIGURE 48 The Geographical Distribution of the Referral Farms of 39 Cattle with Benign Urinary Bladder Neoplasia. (Epidemiology case numbers: E68-E71, E74, E76, E78, E79, E81-E84, E166, E167, E169, E170, E173, E174, E178, E180, E182, E184, E188-E203 and E205, Appendix 4).

- ● Benign urinary bladder neoplasia
- ■ Benign and malignant urinary bladder neoplasia
- ◆ Benign urinary bladder neoplasia and upper alimentary squamous cell carcinoma
- ✸ Benign and malignant urinary bladder neoplasia and upper alimentary squamous cell carcinoma
- \+ Benign urinary bladder neoplasia, upper alimentary squamous cell carcinoma and intestinal adenocarcinoma

nil infestation. The chi-squared test (Table 50) indicates that there is a highly significant association ($p = <0.001$) between these neoplasms and the severity of bracken infestation in the referral area. When only animals with benign urinary bladder neoplasms in the absence of any malignancy are considered there is little change in the proportions referred from areas of severe, moderate and light bracken infestation (56%, 31% and 13% respectively) and the association between these neoplasms and the severity of bracken infestation in the referral area remains highly significant (Table 50).

Other malignant neoplasms in adult animals (OMN)

The geographical distribution of the referral farms of 49 adult animals affected by OMN is recorded in Figure 49 and Table 45. The majority of the animals were referred from the western and central regions both of which accounted for 40.4 per cent of the cases. The proportion of animals with OMN from the western and central regions corresponded more closely to the proportion of all adult admissions from these regions (24.7% and 55.2% respectively) than was found with any of the alimentary or urinary bladder neoplasms previously described. In addition, when the 13 animals with OMN which also had UAP are excluded, the proportion of animals with OMN from each region approximated closely to that for all adult admissions (Table 45).

When the geographical distribution of the referral farms of adult animals with OMN (Figure 49) is compared with

TABLE 50

The Severity of Bracken Infestation in the Areas of the Referral Farms of Animals with Benign Urinary Bladder Neoplasia Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
BUBN (All Cases)	24	11	4	<0.001
BUBN (No Malignancy)	9	5	2	<0.001
All Adult Admissions	287	97	594	-

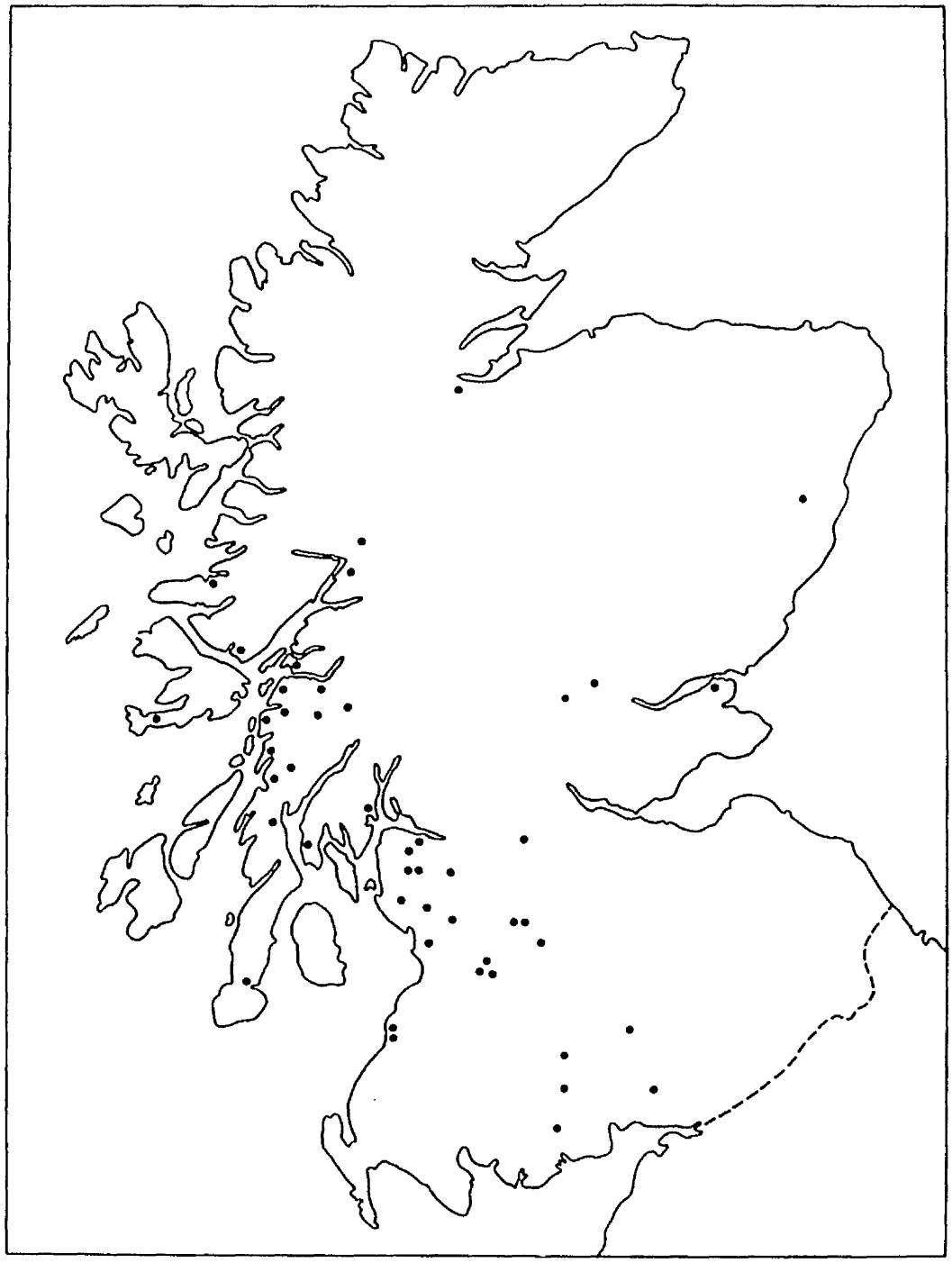


FIGURE 49 The Geographical Distribution of the Referral Farms of 49 Cattle with other Malignant Neoplasms

the geographical distribution of bracken fern (Figure 42) it is found that 37 per cent of the adult animals with OMN were referred from areas of severe bracken infestation, 14 per cent from areas of moderate infestation and 49 per cent from areas of light infestation. Although the proportions of animals with OMN from areas of severe or moderate bracken infestation are slightly greater than those of all adult admissions (29% and 10% respectively) the chi-squared test indicates that there is no association ($p = >0.20$) between these neoplasms and the severity of bracken infestation (Table 51). When only animals affected by OMN in the absence of UAP are considered the proportions from areas of severe, moderate and light infestation (31%, 11% and 58% respectively) are almost identical to the proportions of all adult admissions from these areas (29%, 10% and 61% respectively) and no association ($p = >0.95$) exists between OMN and the severity of bracken infestation in the referral area.

All admissions of immature cattle

The geographical distribution of the referral farms of the 1350 immature cattle admitted from farms in Scotland between 1.9.71 and 31.8.79 is recorded in Figure 50 and Table 45. Eighty-four per cent originated in two regions; the central region (71.4%) and the southern region (13.3%).

When the geographical distribution of the referral farms of all immature admissions are compared with the geographical distribution of bracken fern (Figure 42) it

TABLE 51

The Severity of Bracken Infestation in the Areas of the Referral Farms of Adult Animals Affected by Other Malignant Neoplasms Compared with All Adult Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
OMN (All Cases)	18	7	24	>0.2
OMN (No UAP)	11	4	21	>0.95
All Adult Admissions	287	97	594	-

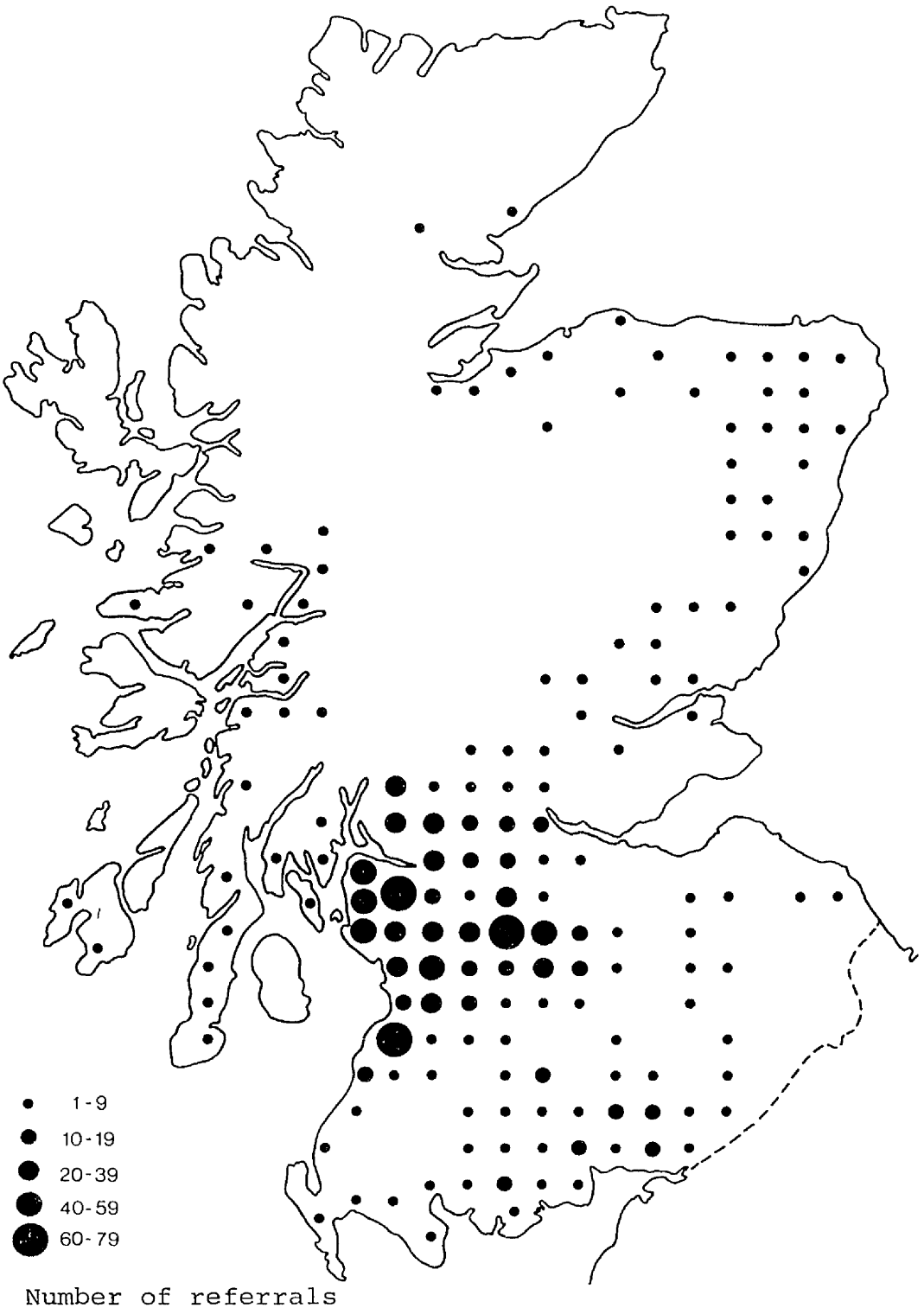


FIGURE 50 The Geographical Distribution of the Referral Farms of 1350 Immature Cattle Admitted during the Period 1.9.71 to 31.8.79.

is found that 81 per cent are situated in areas of light or nil bracken infestation, seven per cent in areas of moderate infestation and 12 per cent in areas of severe infestation.

Malignant neoplasms in immature cattle

The geographical distribution of the referral farms of 76 immature animals affected by malignant neoplasia is recorded in Figure 51and Table 45. As with all immature admissions, the vast majority (81%) of the animals originated in the central and southern regions but, in comparison, the proportion from the central region was smaller (60%) and from the southern region greater (21%). The proportions of affected animals from the northern,western and eastern regions (3%, 8% and 8% respectively) were similar to those for all immature admissions (2%, 8% and 6% respectively).

When the geographical distribution of the referral farms of immature animals affected by malignancies (Figure 51) is compared with the geographical distribution of bracken fern (Figure 42) it is found that 74 per cent were referred from areas of light or nil bracken infestation, nine per cent from areas of moderate infestation and 17 per cent from areas of severe infestation. These proportions do not vary markedly from the respective values obtained for all immature admissions (81%, 7% and 12%), and the chi-squared test indicates that there is no association ($p > 0.20$) between malignant neoplasms in immature animals and the severity of bracken infestation in the referral area (Table 52).

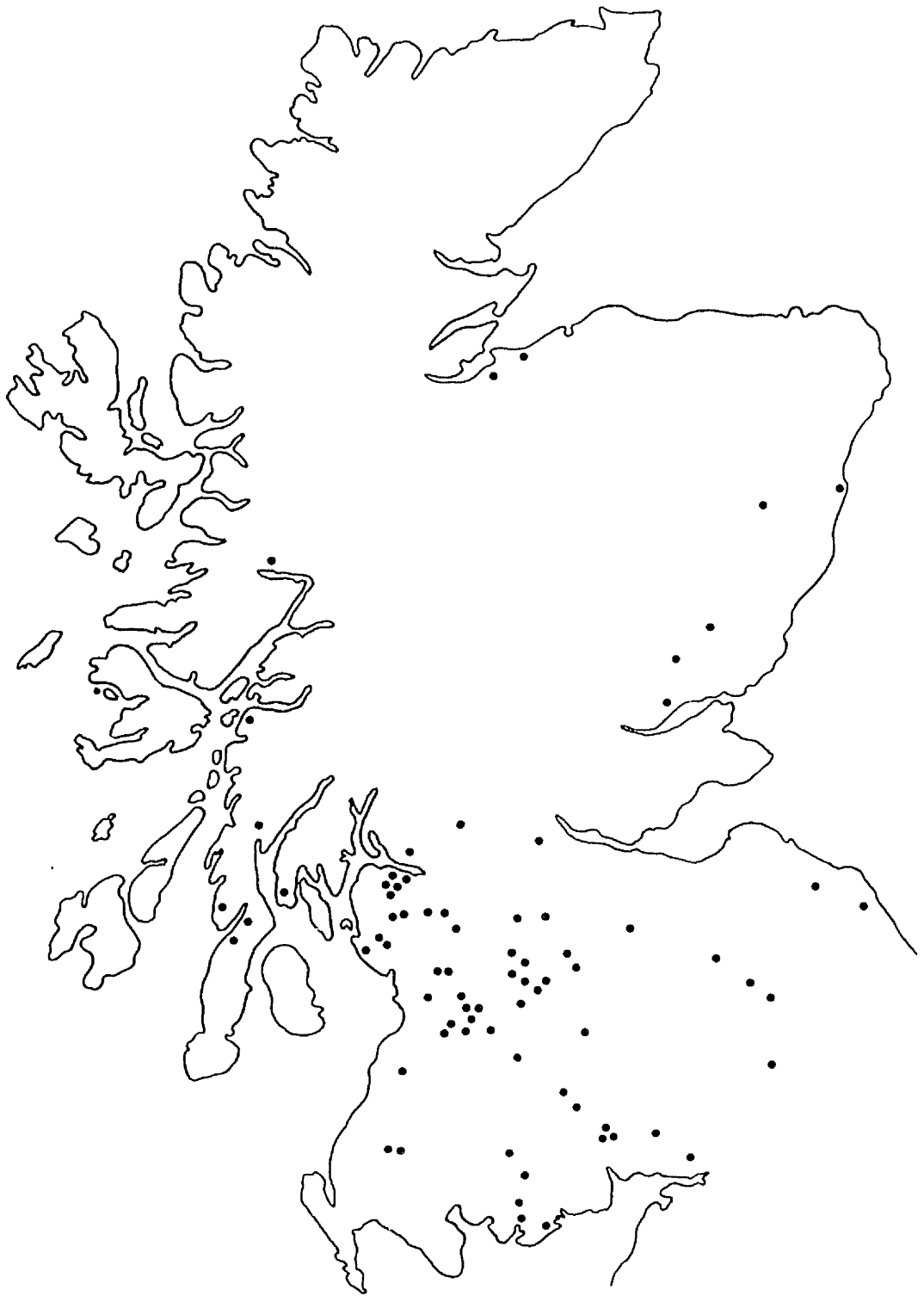


FIGURE 51 The Geographical Distribution of the Referral Farms of 76 Immature Cattle with Malignant Neoplasia

TABLE 52

The Severity of Bracken Infestation in the Areas of the Referral Farms of Immature Animals with Malignant Neoplasia Compared with All Immature Admissions

	Bracken Density			Chi-squared Test Probability (p)
	Severe	Moderate	Light or Nil	
Immature Animals with Malignant Neoplasms	13	7	56	>0.20
All Immature Admissions	158	89	1103	-

Multiple Case Farms

In the course of this study, 16 farms were identified from which two or more adult animals with malignant neoplasms were referred. These 16 multiple case farms which had an average adult population of 1466 cattle accounted for 53 referred animals with a total of 57 different malignancies (Table 53).

Upper alimentary squamous cell carcinoma was identified in animals referred from 14 of the farms with a range of two to seven cases per farm. Intestinal adenocarcinoma, malignant urinary bladder neoplasia, a thyroid carcinoma and a renal haemangiosarcoma were also found in animals referred from six of these 14 farms. However both examples of intestinal adenocarcinoma and two of the six examples of malignant urinary bladder neoplasia were present in animals affected by upper alimentary squamous cell carcinoma.

The referrals from the two remaining multiple case farms were, from one, three animals with malignant urinary bladder neoplasia, and from the other, one animal with malignant urinary bladder neoplasia and one with a bronchial carcinoma.

In addition, upper alimentary papillomas and benign urinary bladder neoplasms were found both in animals with and without malignancies referred from the 16 multiple case farms. Upper alimentary papillomas were found in 49 of the 53 animals with malignant neoplasms and in a further 22 referred

TABLE 53

Numbers and Types of Malignant Neoplasms in Cattle Referred from Multiple Case Farms

Farm	Adult Herd Size	Number of Animals with Malignant Neoplasms	Neoplasms			
			UASCC	IAC	MUBN	OMN
Drum	42	4	3			1
Inveryne	40	3	3			
Barrahormaid	100	8	7*		2*	
Borrodale	100	2	2*		1*	
Carse	32	4	4			
Ardnamurchan	420	2	2			
Brenfield	80	3	2**	1**		1
Kilmichaelbeg	18	4	4			
Ardmarnock	75	4	4**	1**		
Gallachoille	26	5	2		3	
Upper Largie	94	3	3			
Swordle	50	2	2			
Leukary	30	2	2			
Balnalachlan	69	2	2			
Ederline	150	2			1	1
Rainton	140	3			3	

* One animal with both UASCC and MUBN

** One animal with both UASCC and IAC

animals in which no malignancy was present (Table 54). Similarly benign urinary bladder neoplasia was found in eight animals with malignant neoplasms and a further five referrals in which there was no malignancy (Table 54). All these latter five cases of benign urinary bladder neoplasia also had upper alimentary papillomas.

The geographical distribution of the multiple case farms is recorded in Figure 52 and it is evident that the majority are concentrated in a relatively localised area and that all are situated in areas of severe bracken infestation (see Figure 42).

TABLE 54

Numbers of Animals with Upper Alimentary Papillomas and Benign Urinary Bladder Neoplasms referred from Multiple Case Farms

Farm	No. of Animals with Malignant Neoplasms	No. of Animals with Upper Alimentary Papillomas		No. of Animals with Benign Urinary Bladder Neoplasms	
		Malignancy present	No Malignancy	Malignancy present	No Malignancy
Drum	4	4	3		
Inveryne	3	3			
Barrahormaid	8	8	8		1
Borrodale	2	2		1	
Carse	4	4		1	
Ardnamurchan	2	2			
Brenfield	3	3	3		2
Kilmichaelbeg	4	4			
Ardmarnock	4	4	4	1	
Gallachoille	5	5	3	2	2
Upper Largie	3	3			
Swordle	2	2			
Leukary	2	2			
Balnalachlan	2	1			
Ederline	2		1	1	
Rainton	3	2		2	

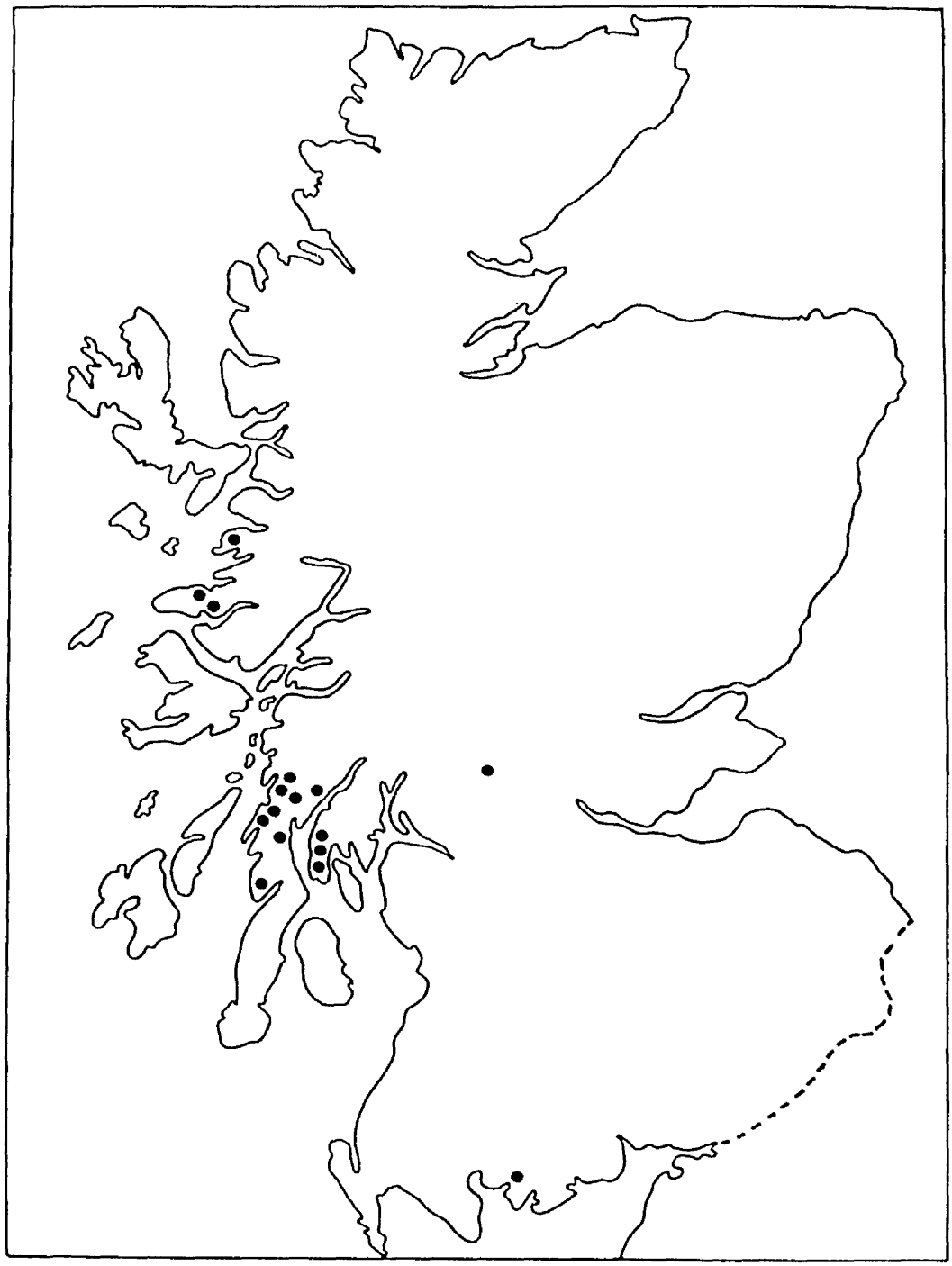


FIGURE 52

The geographical distribution of farms from which multiple cases of malignant neoplasia were referred.

DISCUSSION

Comparison of the geographical distributions of the referral forms of adult cattle with neoplasia and all adult admissions during the same period demonstrated that the distribution pattern of several individual neoplasms did not accord with that of all adult admissions. The origins of animals affected by upper alimentary squamous cell carcinoma, upper alimentary papillomas, urinary bladder neoplasia and intestinal adenocarcinoma tended to be confined to specific areas whereas other malignant neoplasms in adult cattle were widely distributed, in a pattern which approximated that of all adult admissions. Similarly, the origins of immature cattle with malignant neoplasms, principally lymphosarcoma, had a widespread distribution which reflected the pattern of all immature admissions.

The geographical distribution of the origins of cattle with these alimentary and urinary bladder neoplasms is reflected in the geographical distribution of bracken fern and it has been demonstrated that a highly significant association exists between upper alimentary squamous cell carcinoma, upper alimentary papillomas and malignant and benign urinary bladder neoplasia and the severity of bracken infestation in the area from which affected animals were referred. This association is also apparent with respect to intestinal adenocarcinoma but, as there were insufficient cases in which intestinal adenocarcinoma was present in the absence of either upper alimentary or urinary bladder malignancies, it is not possible

to demonstrate whether the association is maintained when intestinal adenocarcinoma is examined in isolation. In contrast there is no evidence of a relationship between bracken fern and other malignant neoplasms of adult cattle or malignant neoplasms, principally lymphosarcoma, of immature animals.

The role of bracken fern in the aetiology of bovine urinary bladder neoplasia is generally accepted but an association between bracken fern and the occurrence of upper alimentary squamous cell carcinoma has only been reported by workers in Brazil (Dobereiner and others, 1967; Campos Neto and others, 1975). In addition, doubt has been cast on the significance of this association by Plowright and others, (1971) who claimed that in the area of Kenya where they performed their studies there was no evidence to implicate bracken fern in the aetiology of upper alimentary squamous cell carcinoma. The present study provides substantial support for the premiss that there is an association between upper alimentary squamous cell carcinoma and the presence of bracken infestation in areas where affected animals are found.

The evidence that there is also a highly significant association between upper alimentary papillomas in adult animals, including in the absence of any malignancy, and the severity of bracken infestation in the area from which affected animals were referred has particular interest. It appears to conflict with the findings of both Thorsen and others, (1974), who reported that the cattle which they found with upper alimentary papillomas "originated from widely separated

geographical areas (in Kenya) with varying climatic conditions and vegetation", and Jarrett and others, (1978b), who specifically stated that "the great majority (of animal with upper alimentary papillomas whose origins they could trace) did not come from upland farms with bracken infestation". These differences are considered in more detail at a later stage, in light of the results described in the following two sections.

In addition to the demonstration that upper alimentary squamous cell carcinoma, upper alimentary papillomas and benign and malignant urinary bladder neoplasms have similar geographical distributions, which are related to the geographical distribution of bracken fern, it is evident from the identification of multiple case farms that these neoplasms occur simultaneously with high frequency amongst small distinct populations of cattle. This would tend to suggest that a common factor(s) could be involved in the aetiology of these neoplasms and in the following two sections the possible role of bracken fern as a common factor is examined in greater detail.

CLINICAL AND EPIDEMIOLOGICAL STUDIES OF BOVINE NEOPLASIA

TWO VOLUMES

VOLUME 2

by

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SECTION III

A CASE CONTROLLED STUDY OF ALIMENTARY AND URINARY BLADDER NEOPLASIA

INTRODUCTION

Once a statistical association between a characteristic and a disease has been observed in a population it is usual to perform a case-control study in which a group of animals which have the disease (the cases) are compared with those which do not (the controls) in order to confirm whether the association is also present within individuals (Lilienfield, 1976). In Section II an association was demonstrated between the geographical distribution of bracken fern and the prevalence of upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia. In the following section a retrospective case control study will be described with the principal purpose of confirming this association.

MATERIALS AND METHODS

(1) Selection of Animals

The animals considered in this section comprise two main groups: (i) Adult cattle affected by neoplasia as described in Chapters 1 and 2, for which a farm of origin was known and for which it was possible to obtain the relevant epidemiological data. (ii) A control group of cattle aged

three years and greater which were pathologically confirmed to be unaffected by neoplasia. This latter group was composed of all such animals admitted to the Medicine Department of the Veterinary Hospital between 1.4.76 and 12.1.80 for which epidemiological data could be obtained. The only other instance in which an animal was excluded was when more than one animal had been referred from an individual farm during this period,in which case only the first referred animal was included in the control group.

With few exceptions the data relating to animals affected by neoplasia was obtained from the owners during visits to the referral farms. Otherwise the data was obtained by communication with owners via letter or telephone. The data relating to each individual animal is recorded in Appendix 4.

(2) Statistical Methods

The statistical methods used were the chi-squared test and the Fisher exact probability test (Siegel, 1956). The Fisher exact probability test was only used in appropriate situations when numbers were insufficient for the chi-squared test to give reliable results.

Prior to comparison of data relating to a specific neoplasm and that relating to the control group it was considered appropriate to standardise the two groups as far as possible with regard to age. Thus for any given analysis control animals which were younger than the minimum age of animal in which the specific neoplasm under consideration

was identified were excluded. In addition, the small number of control animals aged in excess of 14 years resulted in poor matching of controls to cases above this age, and thus all analyses exclude cases and controls greater than 14 years old.

(3) Descriptive Terms

There are several descriptive terms in use in this section which require definition. Severity of bracken infestation is described as light, moderate or severe. Light infestation is applied where less than five per cent of the pastures are bracken infested, moderate infestation where between five and 19 per cent of the pastures are bracken infested and severe where 20 per cent or more of the pastures are bracken infested. The proportion of bracken infested pastures on referral farms is recorded for each animal in Appendix 4 and typical examples of moderate and severe bracken infestation are shown in Figures 53 and 54 respectively.

The recording of an incident of acute bracken poisoning on a farm implies that at least one clinical case of acute bracken poisoning had been observed on the farm during the 15 year period prior to the referral of the animal included in this study. The confirmation of clinical cases was made by the author or the general practitioner of the farm concerned in the majority of cases, but in the remainder the data relating to incidents of acute bracken poisoning was

FIGURE 53

Moderate bracken infestation of a pasture with bracken infestation mainly confined to the field margins.

FIGURE 54

Severe bracken infestation of a pasture.

based on information supplied by the owner. The class of animals involved in incidents of acute bracken poisoning on referral farms and the source of information is recorded in Appendix 4.

RESULTS

Upper Alimentary Squamous Cell Carcinoma (UASCC)

Seventy (97%) of 72 of the animals affected by UASCC were referred from bracken infested farms which is in marked contrast with the similarly aged control animals of which only 44 per cent were referred from bracken infested farms (Table 55). The chi-squared test indicates that there is a highly significant association ($p = < 0.001$) between UASCC and the presence of bracken fern on the referral farm.

Very similar results are obtained when only animals with UASCC but in the absence of any other malignancy are examined (Table 55). The chi-squared test indicates that there is a highly significant association ($p = < 0.001$) between UASCC in the absence of any other malignancy and the presence of bracken fern on the referral farm.

This highly significant association is also evident when only animals which were born on the farm from which they were referred (i.e. "home bred" animals) are considered (Table 55). Every home bred animal affected by UASCC, irrespective of the presence or absence of another malignancy, was referred from a bracken infested farm whereas only 28 per cent of the referral farms of the similarly aged control animals were infested by bracken fern (Table 55).

In most instances, the referral farms of animals with UASCC which were not born on the farm from which they were

TABLE 55

The Bracken Status of Referral Farms of Animals with Upper Alimentary Squamous Cell Carcinoma (UASCC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought in Animals*

	Bracken Status of Referral Farm		Chi Squared Test
	Bracken Absent	Bracken Present	Probability (p)
UASCC (All Cases)	2	70	<0.001
UASCC (No Other Malignancy)	2	58	<0.001
Controls	31	24	-

b) Home Bred Animals Only *

	Bracken Status of Referral Farm		Chi Squared Test
	Bracken Absent	Bracken Present	Probability (p)
UASCC (All Cases)	0	46	<0.001
UASCC (No Other Malignancy)	0	37	<0.001
Controls	23	9	-

* Excluding animals aged less than seven years and greater than 14 years.

referred (i.e. bought in animals) were also bracken infested. Only two of the bought in animals were referred from farms which were bracken free but in both cases the animals were purchased and moved onto the referral farm when adult, aged seven and over ten years respectively, and thereafter were referred to the Veterinary Hospital within three years. Unfortunately, no information was available in either case as to their previous origins.

Fourteen of the bought in animals which were referred from bracken infested farms were purchased and moved to their referral farm when less than three years of age. Thus all these animals had been on bracken infested farms for between five and 13 years. In addition, it was possible to ascertain whether bracken fern was present on the farms on which four of these animals were born and from which they had subsequently been purchased. In each case the farm had bracken infested pastures.

The remaining ten animals were purchased and moved to their referral farms when aged between three and 14 years and on these farms had been exposed to bracken infested pastures until their referral, one to 11 years later. The bracken status of the farms from which six of these animals had been purchased and on which they were also born was ascertained and,in each case, bracken infestation was present.

The severity of bracken infestation on the farms from which animals affected by UASCC were referred is compared with that on the referral farms of similarly aged control animals in Table 56. Eighty-six per cent of all cases of UASCC were referred from farms with moderate or severe bracken infestation compared with only 15 per cent

TABLE 56

The Severity of Bracken Infestation on Referral Farms of Animals with Upper Alimentary Squamous Cell Carcinoma (UASCC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test
	Nil	Light	Moderate	Severe	Probability (p)
UASCC (All Cases)	2	8	26	36	<0.001
UASCC (No Other Malignancy)	2	6	20	30	<0.001
Controls	31	16	3	5	-

b) Home Bred Animals Only*

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test
	Nil	Light	Moderate	Severe	Probability (p)
UASCC (All Cases)	0	5	16	25	<0.001
UASCC (No Other Malignancy)	0	4	12	21	<0.001
Controls	23	5	2	2	-

* Excluding animals aged less than seven years and greater than 14 years.

of the control animals. The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between UASCC and the severity of bracken infestation on the referral farm. This association remains highly significant ($p = <0.001$) when only animals with UASCC in the absence of any other malignancy or when only home bred animals are considered (Table 56).

Incidents of acute bracken poisoning had been recorded on the referral farms of 60 per cent of the animals affected by UASCC compared with only 17 per cent of the bracken infested referral farms of similarly aged control animals (Table 57). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between UASCC and the occurrence of acute bracken poisoning on the referral farm. This highly significant association is maintained when only animals with UASCC in the absence of any other malignancy are considered (Table 57). Similar results were obtained for home bred animals using the Fisher exact probability test which indicated that there is a significant association between all home bred animals affected by upper alimentary squamous cell carcinoma ($p = 0.016$) or only those cases without any other malignancy ($p = 0.012$) and the occurrence of acute bracken poisoning on the referral farm (Table 57).

TABLE 57

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with Upper Alimentary Squamous Cell Carcinoma (UASCC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Acute Bracken Poisoning on Bracken Infested Farms		Chi-squared Test Probability (p)
	Not Recorded	Recorded	
UASCC (All Cases)	28	42	<0.001
UASCC (No Other Malignancy)	22	36	<0.001
Controls	20	4	-

b) Home Bred Animals Only *

	Acute Bracken Poisoning on Bracken Infested Farms		Fisher Exact Probability Test Probability (p)
	Not Recorded	Recorded	
UASCC (All Cases)	15	31	0.016
UASCC (No Other Malignancy)	11	26	0.012
Controls	7	2	-

* Excluding animals aged less than seven years and greater than 14 years.

Upper Alimentary Papillomas (UAP)

One hundred and fifty (93%) of the 161 animals affected by UAP were referred from bracken infested farms which is in marked contrast with the similarly aged control animals, of which only 40 per cent were referred from bracken infested farms (Table 58). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between UAP and the presence of bracken fern on the referral farm. Almost identical results are obtained when only animals affected by UAP in the absence of any malignancy are considered (Table 58) and a highly significant association between these neoplasms and the presence of bracken on the referral farm is shown by the chi-squared test ($p = <0.001$).

This highly significant association is also evident when only home bred animals are considered (Table 58). Ninety-six per cent of all home bred animals affected by upper alimentary papillomas and 93 per cent of home bred animals affected by UAP in the absence of any malignancy were referred from bracken infested farms, whereas only 27 per cent of the similarly aged control animals were referred from such farms (Table 58).

The three home bred animals with UAP in the absence of any malignancy which were referred from farms on which no bracken was present were aged three, four and six years. The youngest was a beef animal referred from the southern region and the other two were dairy animals referred from the southern and central regions. The single home bred animal with UAP which was affected by a malignancy and was referred from a bracken free farm, also in the

TABLE 58

The Bracken Status of Referral Farms of Animals with Upper Alimentary Papillomas (UAP) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
UAP (All Cases)	11	150	<0.001
UAP (No malignancy)	6	61	<0.001
Controls	56	38	-

b) Home Bred Animals Only *

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
UAP (All Cases)	4	96	<0.001
UAP (No malignancy)	3	36	<0.001
Controls	43	16	-

* Excluding animals aged less than three years and greater than 14 years.

southern region, had an osteosarcoma of the pelvis and was aged nine years.

Of the seven bought in animals with UAP which were referred from bracken free farms, four had malignancies of which two were affected by upper alimentary squamous cell carcinoma and two by malignant urinary bladder neoplasms. Unfortunately there was no information available as to the previous origins of any of these animals of which six were bought in when aged greater than two and a half years and one, in which there was no malignancy, when aged one month.

The severity of bracken infestation on the farms from which animals affected by UAP were referred is compared with that on the referral farms of similarly aged control animals in Table 59. Eighty-four per cent of all animals with UAP were referred from farms with moderate or severe bracken infestation compared with only 15 per cent of the control animals. The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between UAP and the severity of bracken infestation on the referral farm. This association remains highly significant ($p = <0.001$) when only animals with UAP in the absence of any malignancy or when only home bred animals are considered (Table 59).

In addition, it is worthwhile to note that the proportions of home bred animals with UAP in the absence of any malignancy which were referred from moderately and severely bracken infested farms (28 and 56% respectively) are very similar to those of home bred animals with upper

TABLE 59

The Severity of Bracken Infestation on Referral Farms of Animals with Upper Alimentary Papillomas (UAP) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
UAP (All Cases)	11	15	58	77	<0.001
UAP (No Malignancy)	6	7	20	34	<0.001
Controls	56	24	7	7	-

b) Home Bred Animals Only *

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
UAP (All Cases)	4	8	36	52	<0.001
UAP (No Malignancy)	3	3	11	22	<0.001
Controls	43	11	2	3	-

* Excluding animals aged less than three years and greater than 14 years.

alimentary squamous cell carcinoma in the absence of any other malignancy (32 and 57% respectively).

Incidents of acute bracken poisoning had been recorded on the referral farms of 59 per cent of the animals affected by UAP compared with only 16 per cent of the bracken infested referral farms of similarly aged control animals (Table 60). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between UAP and the occurrence of acute bracken poisoning on the referral farm. This highly significant association is maintained when only animals with UAP in the absence of any malignancy or when only home bred animals are considered (Table 60).

TABLE 60

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with Upper Alimentary Papillomas (UAP) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared Test Probability (p)
	Not Recorded	Recorded	
UAP (All Cases)	62	88	<0.001
UAP (No Malignancy)	23	38	<0.001
Controls	32	6	-

b) Home Bred Animals Only *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared Test Probability (p)
	Not Recorded	Recorded	
UAP (All Cases)	33	63	<0.001
UAP (No Malignancy)	10	26	<0.001
Controls	13	3	-

* Excluding animals aged less than three years and greater than 14 years.

Intestinal Adenocarcinoma (IAC)

Eleven (92%) of the 12 animals affected by IAC were referred from bracken infested farms which is in marked contrast with the similarly aged control animals of which only 42 per cent were referred from bracken infested farms (Table 61). The chi-squared test indicates that there is a significant association ($p = <0.01$) between IAC and the presence of bracken fern on the referral farm.

Examination of the animals with IAC in the absence of any other malignancy (Table 61) reveals that five of the six animals were referred from bracken infested farms. However, the numbers of animals are considered insufficient to allow reliable statistical analysis as to whether there is any association between IAC in the absence of any other malignancy and the presence of bracken fern on the referral farm.

When only home bred animals are considered it is found that every animal affected by IAC was referred from a bracken infested farm whereas only 31 per cent of the referral farms of similarly aged control animals were infested by bracken fern. The Fisher exact probability test indicates that there is a significant association ($p = 0.003$) between IAC in home bred animals and the presence of bracken fern on the referral farm (Table 61).

However, it is not possible to demonstrate whether this association is maintained for home bred animals with IAC in the absence of any other malignancy as

TABLE 61

The Bracken Status of Referral Farms of Animals with Intestinal Adenocarcinoma (IAC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
IAC (All Cases)	1	11	<0.01
IAC (No Other Malignancy)	1	5	**
Controls	46	33	-

b) Home Bred Animals Only*

	Bracken Status of Referral Farm		Fisher Exact Probability Test Probability (p)
	Bracken Absent	Bracken Present	
IAC (All Cases)	0	8	0.003
IAC (No Other Malignancy)	0	4	**
Controls	33	15	-

* Excluding animals aged less than five years and greater than 11 years

** Numbers insufficient for analysis

although all four such animals were referred from bracken infested farms this small number is considered insufficient to allow reliable statistical analysis.

The severity of bracken infestation on the farms from which animals affected by IAC were referred is compared with that on the referral farms of similarly aged control animals in Table 62. However there are insufficient numbers of animals with IAC to allow reliable statistical analysis as to whether there is any association between IAC and the severity of bracken infestation on the referral farm.

Incidents of acute bracken poisoning had been recorded on the referral farms of 55 per cent of the animals affected by IAC compared with only 15 per cent of the bracken infested referral farms of similarly aged control animals (Table 63). The Fisher exact probability test indicates that there is a significant association ($p = 0.016$) between IAC and the occurrence of acute bracken poisoning on the referral farm. However due to the small number of animals it is not possible to demonstrate whether this association is maintained in animals with IAC in the absence of any other malignancy.

In contrast, when only home bred animals with IAC are considered (Table 63) there is no significant association ($p = 0.156$) between IAC and the occurrence of acute bracken poisoning on the referral farm. As before, it is not possible, due to the small number of animals, to demonstrate whether the association is not present in home bred animals with IAC in the absence of any other malignancy.

TABLE 62

The Severity of Bracken Infestation on Referral Farms of Animals with Intestinal Adenocarcinoma (IAC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
IAC (All Cases)	1	2	6	3	**
IAC (No Other Malignancy)	1	1	4	0	**
Controls	46	23	5	5	-

b) Home Bred Animals Only *

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
IAC (All Cases)	0	1	5	2	**
IAC (No Other Malignancy)	0	1	3	0	**
Controls	33	11	2	2	-

* Excluding animals aged less than five years and greater than 11 years.

** Numbers insufficient for analysis.

TABLE 63

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with Intestinal Adenocarcinoma (IAC) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Fisher Exact Probability Test
	Not Recorded	Recorded	Probability (p)
IAC (All Cases)	5	6	0.0162
IAC (No Other Malignancy)	3	2	**
Controls	28	5	-

b) Home Bred Animals Only*

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Fisher Exact Probability Test
	Not Recorded	Recorded	Probability (p)
IAC (All Cases)	4	4	0.1556
IAC (No Other Malignancy)	3	1	**
Controls	12	3	-

* Excluding animals aged less than five years and greater than 11 years.

** Numbers insufficient for analysis.

Malignant Urinary Bladder Neoplasms (MUBN)

Twenty-five (93%) of the 27 animals affected by MUBN were referred from bracken infested farms which is in marked contrast with the similarly aged control animals of which only 40 per cent were referred from bracken infested farms (Table 64). The chi-squared test indicates that there is a highly significant association between MUBN and the presence of bracken fern on the referral farm. Very similar results are obtained when only animals with MUBN in the absence of any other malignancy are examined (Table 64). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between MUBN in the absence of any other malignancy and the presence of bracken fern on the referral farm.

This highly significant association is also evident when only home bred animals are considered (Table 64). Every animal affected by MUBN was referred from a bracken infested farm whereas only 27 per cent of the referral farms of the similarly aged control animals were infested by bracken fern.

In most instances, the referral farms of bought in animals with MUBN were also bracken infested. Two of the bought in animals were referred from farms which were bracken free but in both cases the animals were purchased and moved onto the referral farm when aged over five years and thereafter were referred to the Veterinary Hospital within six months. Unfortunately, no information was available in either case as to their previous origins. The

TABLE 64

The Bracken Status of Referral Farms of Animals with Malignant Urinary Bladder Neoplasia (MUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Bracken Status of Referral Farms		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
MUBN (All Cases)	2	25	<0.001
MUBN (No Other Malignancy)	2	17	<0.001
Controls	56	38	-

b) Home Bred Animals Only *

	Bracken Status of Referral Farms		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
MUBN (All Cases)	0	21	<0.001
MUBN (No Other Malignancy)	0	15	<0.001
Controls	43	16	-

* Excluding animals aged less than three years and greater than 14 years.

remaining four bought in animals of which one was affected by upper alimentary squamous cell carcinoma were moved to their referral farms when aged between one month and four years. There was no information available regarding the bracken status of their previous origin but all four cases had access to bracken on their referral farms for between three and 12 years.

The severity of bracken infestation on the farms from which animals affected by MUBN were referred is compared with that on the referral farms of similarly aged control animals in Table 65. Due to the relatively small numbers of animals involved it is necessary to combine the groups from farms with light and moderate infestation in order that the chi-squared test gives reliable results. Fifty-two per cent of the animals with MUBN were referred from farms with severe bracken infestation compared with only seven per cent of the control animals and the chi-squared test indicates that there is a highly significant association ($p = <0.001$) between MUBN and the severity of bracken infestation on the referral farm. This association remains highly significant ($p = <0.001$) when only animals with MUBN in the absence of any other malignancy or when only home bred animals are considered (Table 65). However, in the specific case of the home bred animals in the absence of any other malignancy the results of the chi-squared test are unreliable (Table 65).

Incidents of acute bracken poisoning had been recorded on the referral farms of 56 per cent of the animals affected by MUBN compared with only 16 per cent of

TABLE 65

The Severity of Bracken Infestation on Referral Farms of Animals with Malignant Urinary Bladder Neoplasia (MUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test Probability (p)
	Nil	Light	Moderate	Severe	
MUBN (All Cases)	2	1	10	14	<0.001 **
MUBN (No Other Malignancy)	2	0	8	9	<0.001 **
Controls	56	24	7	7	-

b) Home Bred Animals Only *

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test Probability (p)
	Nil	Light	Moderate	Severe	
MUBN (All Cases)	0	1	10	10	<0.001 **
MUBN (No Other Malignancy)	0	0	8	7	***
Controls	43	11	2	3	-

* Excluding animals aged less than three years and greater than 14 years.

** Light and Moderate colums combined for chi-squared test.

*** Chi-squared test unreliable. Two cells with expected frequencies less than five.

the bracken infested referral farms of similarly aged control animals (Table 66). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between MUBN and the occurrence of acute bracken poisoning on the referral farm. A significant association is maintained when only animals with MUBN in the absence of any other malignancy or when only home bred animals are considered (Table 66).

TABLE 66

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with Malignant Urinary Bladder Neoplasia (MUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared[a] or Fisher Exact Probability[b] Tests
	Not Recorded	Recorded	Probability (p)
MUBN (All Cases)	11	14	<0.001[a]
MUBN (No Other Malignancy)	7	10	0.002[b]
Controls	32	6	-

b) Home Bred Animals Only *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared Test
	Not Recorded	Recorded	Probability (p)
MUBN (All Cases)	9	12	<0.02
MUBN (No Other Malignancy)	6	9	<0.02
Controls	13	3	-

* Excluding animals aged less than three years and greater than 14 years.

Benign Urinary Bladder Neoplasms (BUBN)

Thirty-five (97%) of the 36 animals affected by BUBN were referred from bracken infested farms which is in marked contrast with the similarly aged control animals of which only 41 per cent were referred from bracken infested farms (Table 67). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between BUBN and the presence of bracken fern on the referral farm. Very similar results are obtained when only animals with BUBN in the absence of any malignancy are examined (Table 67). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between BUBN in the absence of any malignancy and the presence of bracken fern on the referral farm.

This highly significant association is also evident when only home bred animals are considered (Table 67). Every animal affected by BUBN was referred from a bracken infested farm whereas only 32 per cent of the referral farms of similarly aged control animals were infested by bracken fern.

In the vast majority of cases, the referral farms of bought in animals with BUBN were also bracken infested. The single bought in animal which was referred from a farm which was bracken free was purchased and moved onto that farm when aged over five years, developed haematuria three weeks later and was only kept on the referral farm for five months before being sent to the Veterinary Hospital. This animal was also affected by malignant urinary bladder

TABLE 67

The Bracken Status of Referral Farms of Animals with Benign Urinary Bladder Neoplasia (BUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
BUBN (All Cases)	1	35	<0.001
BUBN (No Malignancy)	0	16	<0.001
Controls	44	31	-

b) Home Bred Animals Only *

	Bracken Status of Referral Farm		Chi-squared[a] or Fisher Exact Probability[b] Tests Probability (p)
	Bracken Absent	Bracken Present	
BUBN (All Cases)	0	20	<0.001[a]
BUBN (No Malignancy)	0	9	0.0002[b]
Controls	32	15	-

* Excluding animals aged less than six years and greater than 14 years.

neoplasia but, unfortunately, no information was available as to its previous origins. The remaining 15 bought in animals, eight of which also had malignancies, were moved to their referral farms when aged between one month and five years and on these farms had been exposed to bracken infested pastures for between four and 12 years. In the four cases in which it was possible to investigate the previous origins of the animal, the farm was found in each case to have bracken infested pastures.

The severity of bracken infestation on the farms from which animals affected by BUBN were referred is compared with that on the referral farms of similarly aged control animals in Table 68. Fifty-six per cent of the animals with BUBN were referred from farms with severe bracken infestation compared with only eight per cent of the control animals and the chi-squared test indicates that there is a highly significant association ($p = <0.001$) between BUBN and the severity of bracken infestation on the referral farm. When only animals with BUBN in the absence of any malignancy and when only home bred animals are considered it is necessary due to the relatively small numbers of animals to combine the groups from farms with low and medium infestation in order that the chi-squared test gives reliable results. The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between BUBN in the absence of any malignancy and the severity of bracken infestation. When only home bred animals are considered this association remains highly significant ($p = <0.001$) if all animals with BUBN are

TABLE 68

The Severity of Bracken Infestation on Referral Farms of Animals with Benign Urinary Bladder Neoplasia (BUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test Probability (p)
	Nil	Light	Moderate	Severe	
BUBN (All Cases)	1	3	12	20	<0.001
BUBN (No Malignancy)	0	2	2	12	<0.001 **
Controls	44	21	4	6	

b) Home Bred Animals Only *

	Severity of Bracken Infestation on Referral Farms				Chi-Squared Test Probability (p)
	Nil	Light	Moderate	Severe	
BUBN (All Cases)	0	2	7	11	<0.001 **
BUBN (No Malignancy)	0	2	1	6	***
Controls	32	10	2	3	-

* Excluding animals aged less than six years and greater than 14 years.

** Light and Moderate colums combined with chi-squared test.

*** Chi-squared test unreliable. Two cells with expected frequencies less than five.

included, but when only those animals which do not have any malignancy are considered, the numbers are insufficient for the chi-squared test to give reliable results (Table 68).

Incidents of acute bracken poisoning had been recorded on the referral farms of 69 per cent of the animals affected by BUBN compared with only 16 per cent of the bracken infested referral farms of similarly aged control animals (Table 69). The chi-squared test indicates that there is a highly significant association ($p = <0.001$) between BUBN and the occurrence of acute bracken poisoning on the referral farm. A significant association is maintained when only animals with BUBN in the absence of any malignancy or when only home bred animals are considered (Table 69).

TABLE 69

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with Benign Urinary Bladder Neoplasia (BUBN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared Test
	Not Recorded	Recorded	Probability (p)
BUBN (All Cases)	11	24	<0.001
BUBN (No Malignancy)	5	11	<0.001
Controls	26	5	-

b) Home Bred Animals Only *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Chi-squared[a] or Fisher Exact Probability[b] Test Probability (p)
	Not Recorded	Recorded	
BUBN (All Cases)	7	13	<0.01 [a]
BUBN (No Malignancy)	2	7	0.0088 [b]
Controls	12	3	-

* Excluding animals aged less than six years and greater than 14 years.

Other Malignant Neoplasms (OMN)

Twenty-one (51%) of the 41 animals affected by other malignant neoplasms were referred from bracken infested farms which is a slightly higher proportion than was found in the similarly aged control animals of which 40 per cent were referred from bracken infested farms (Table 70). However the chi-squared test indicates that there is no association between the other malignant neoplasms and the presence of bracken on the referral farm. When only those animals which had other malignant neoplasms in the absence of any upper alimentary papillomas are considered, the proportion referred from bracken infested farms falls to that of the control animals (i.e. 40%) and no association between these neoplasms and the presence of bracken on the referral farm can be demonstrated.

When only home bred animals are considered it is found that 40 per cent of all animals affected by other malignant neoplasms and 30 per cent of animals affected by other malignant neoplasms in the absence of upper alimentary papillomas were referred from bracken infested farms compared with 27 per cent of the control animals (Table 70).

The severity of bracken infestation on the farms from which animals affected by other malignant neoplasms were referred is compared with that on the referral farms of similarly aged control animals in Table 71. Due to the relatively small numbers of animals involved it is necessary to combine the groups from farms with light and moderate infestation in order that the chi-squared test gives reliable

TABLE 70

The Bracken Status of Referral Farms of Animals with Other Malignant Neoplasms (OMN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
OMN (All Cases)	20	21	>0.2
OMN (No UAP)	19	12	>0.8
Controls	56	38	-

b) Home Bred Animals Only*

	Bracken Status of Referral Farm		Chi-squared Test Probability (p)
	Bracken Absent	Bracken Present	
OMN (All Cases)	15	10	>0.2
OMN (No UAP)	14	6	>0.8
Controls	43	16	-

* Excluding animals aged less than three years and greater than 14 years.

TABLE 71

The Severity of Bracken Infestation on Referral Farms of Animals with other Malignant Neoplasms (OMN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
OMN (All Cases)	20	6	9	6	>0.2**
OMN (No UAP)	19	3	5	4	>0.5**
Controls	56	24	7	7	-

b) Home Bred Animals Only*

	Severity of Bracken Infestation on Referral Farms				Chi-squared Test Probability (p)
	Nil	Light	Moderate	Severe	
OMN (All Cases)	15	2	5	3	***
OMN (No UAP)	14	1	3	2	***
Controls	43	11	2	3	-

* Excluding animals aged less than three years and greater than 14 years.

** Light and Moderate columns combined with chi-squared test.

*** Chi-squared test unreliable. Two cells with expected frequencies less than five.

results. Fifteen per cent of the animals with other malignant neoplasms were referred from farms with severe bracken infestation compared with seven per cent of the control animals and the chi-squared test indicates that there is no association ($p = >0.2$) between other malignant neoplasms and the severity of bracken infestation on the referral farm. When only those animals which had other malignant neoplasms in the absence of any upper alimentary papillomas are considered the proportion referred from farms with severe bracken infestation is 13 per cent and no association ($p = <0.5$) between these neoplasms and the severity of bracken infestation can be demonstrated.

When only home bred animals are considered it is found that 12 per cent of all animals with other malignant neoplasms and 10 per cent of animals with other malignant neoplasms in the absence of upper alimentary papillomas were referred from farms with severe bracken infestation compared with five per cent of the control animals (Table 71). However, it is not possible to demonstrate whether any significant difference exists between the affected animals of either group and the control animals as, due to inadequate numbers, the results of the chi-squared test are unreliable.

Incidents of acute bracken poisoning had been recorded on the referral farms of 29 per cent of the animals affected by other malignant neoplasms compared with 16 per cent of the bracken infested referral farms of similarly aged control animals (Table 72). The Fisher

TABLE 72

The Occurrence of Acute Bracken Poisoning on Referral Farms of Animals with other Malignant Neoplasms (OMN) Compared with Control Animals Unaffected by Neoplasia

a) Home Bred and Bought In Animals*

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Fisher Exact Probability Test Probability (p)
	Not Recorded	Recorded	
OMN (All Cases)	15	6	0.2018
OMN (No UAP)	9	3	0.3683
Controls	32	6	-

b) Home Bred Animals Only *

	Acute Bracken Poisoning on Bracken Infested Referral Farms		Fisher Exact Probability Test Probability (p)
	Not Recorded	Recorded	
OMN (All Cases)	7	3	0.4198
OMN (No UAP)	4	2	0.4195
Controls	13	3	-

* Excluding animals aged less than three years and greater than 14 years.

exact probability test indicates that there is no association between other malignant neoplasms and the occurrence of acute bracken poisoning on the referral farm. Similarly no association could be found when only animals with other malignant neoplasms in the absence of upper alimentary papillomas or when only home bred animals are considered (Table 72).

Multiple Case Farms

As recorded in the previous section, 16 multiple case farms were identified from which two or more adult animals with malignant neoplasms were referred. Bracken infestation was found to be present on all of these farms and the degree of bracken infestation tended to be more severe than on farms from which only single cases of upper alimentary squamous cell carcinoma, intestinal adenocarcinoma or malignant urinary bladder neoplasia were referred (Table 73). In addition, incidents of acute bracken poisoning were recorded on a higher proportion of multiple case farms (69%) than single case farms (41%).

DISCUSSION

The results of this case - control study provide further evidence of the close association between exposure to bracken fern and the occurrence of upper alimentary squamous cell carcinoma, upper alimentary papillomas and benign and malignant urinary bladder neoplasia. All the animals affected by upper alimentary squamous cell carcinoma and benign and

TABLE 73 The Severity of Bracken Infestation and the Frequency with which Acute Bracken Poisoning Incidents were Recorded on Multiple Case Farms, Single Case Farms and Control Farms

Farms	Severity of Bracken Infestation				Acute Bracken Poisoning Incidents Recorded (%)
	Nil(%)	Light(%)	Moderate(%)	Severe(%)	
Multiple Case Farms	0	7	40	53	69
Single Case Farms	8	11	48	33	41
Control Farms	60	26	7	7	6

malignant urinary bladder neoplasms, for which a complete life history could be obtained, had been exposed to bracken fern and, in a high proportion of cases, consumption of bracken by cattle on their farms of origin was confirmed by the occurrence of acute bracken poisoning incidents.

In most respects the findings with regard to animals affected by upper alimentary papillomas were similar. However, there was one notable difference in that four animals with upper alimentary papillomas were born and remained throughout their lives on farms on which there was no apparent bracken infestation. Thus, although there is a highly significant association between upper alimentary papillomas and the presence of bracken fern on the referral farms of affected animals, it would appear that exposure to bracken fern is not a prerequisite for the development of these papillomas.

As in the previous section, it was not possible to identify whether or not there is an association between intestinal adenocarcinoma and the presence of bracken fern on the referral farms of affected animals. The results obtained tend to suggest that there may be an association, but it would be necessary to examine larger numbers of animals with intestinal adenocarcinoma, particularly in the absence of other malignancies which have been shown to be closely associated with bracken fern, before any firm conclusions could be drawn.

The group of animals with other malignant neoplasms is comprised of animals with a wide range of different

malignancies and can be regarded as a second control group. The fact that there is no apparent association between this group and bracken fern, even when animals which have upper alimentary papillomas are included, adds further weight to the evidence for an association in the case of the upper alimentary and urinary bladder neoplasms.

Additional support is also engendered by the data relating to multiple case farms in that the severity of bracken infestation and the frequency of acute bracken poisoning incidents is greater on these farms than on single case farms.

In summary, there is considerable evidence that a close association exists between the occurrence of upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia and exposure to bracken fern. However, in the case of upper alimentary papillomas, exposure to bracken fern does not appear to be a prerequisite for their development.

SECTION IV

A STUDY OF THE PREVALENCE OF UPPER ALIMENTARY PAPILLOMAS IN CATTLE ON BRACKEN INFESTED AND BRACKEN-FREE FARMS

INTRODUCTION

Despite the apparent frequency with which papillomas occur in the upper alimentary tract of cattle (Cotchin, 1957; Nieberle and Cohrs, 1967) the information available with regard to their epidemiology is extremely limited. Similarly, there have been few attempts to identify the aetiology of upper alimentary papillomas although a virus has recently been implicated in studies performed by Jarrett and others (1978b).

However, in the course of this study a highly significant association has been identified between the presence of upper alimentary papillomas and bracken infestation on the referral farms of affected animals (see Chapter 3, Section III). In order to further investigate the possible role of bracken fern in the aetiology of upper alimentary papillomas the following study of their prevalence on bracken infested and bracken-free farms was conducted.

MATERIALS AND METHODS

(1) Selection of Farms

Twelve bracken infested farms and five bracken-free farms were selected. Animals affected by various malignant neoplasms had been referred from nine of the bracken infested farms but no cases of malignant neoplasia had been referred from the remaining three (Table 74). Incidents of acute bracken poisoning had been recorded on nine of the bracken infested farms. No cases of malignant neoplasia had been referred from any of the bracken-free farms.

(2) Examination of Animals

On each farm, the hard and soft palate of all available animals aged 18 months and older was examined for the presence of papillomas by palpation. In any cases in which there was doubt as to their presence or absence a visual examination was performed using a torch for illumination and, when necessary, a mouth gag to facilitate inspection of the oral cavity.

(3) Statistical Methods

The statistical method used was the coefficient of correlation (Bishop, 1971). Unless otherwise stated, when a difference is described as significant, this implies that the probability of its resulting from chance is less than two percent ($p = < 0.02$) and when a difference is described as highly significant this implies that the probability of its resulting from chance is less than 0.1 percent ($p =< 0.001$)

TABLE 74 The Farms on which Examinations for the Presence of Palatine Papillomas were Performed

Farm	No. Animals Examined	Types/Nos. of Malignant Neoplasms in Referred Adult Animals	Incident(s) of Acute Bracken Poisoning	Severity of Bracken Infestation
BR1	31	UASCC (Case No. E13) ocular squamous cell carcinoma (Case No. E243)	-	Moderate (13% of pastures)
BR2	30	UASCC (Case Nos. E11,E23,E43,E70)	+	Severe (20% of pastures)
BR3	28	None	+	Moderate (16% of pastures)
BR4	22	UASCC (Case Nos.E1,E7,E79) Thyroid carcinoma (Case No. E220)	+	Moderate (13% of pastures)
BR5	30	MUBN (Case No. E181)	+	Severe (23% of pastures)
BR6	36	None	+	Severe (33% of pastures)
BR7	167	UASCC (Case Nos. E60, E61)	+	Severe (25% of pastures)
BR8	101	Lymphosarcoma (Case No. E211)	-	Severe (70% of pastures)
BR9	72	Squamous carcinoma of small intestine (Case No. E213)	+	Severe (42% of pastures)
BR10	26	UASCC (Case Nos. E28,E29) MUBN (Case No. E180, E182, E185)	+	Severe (60% of pastures)
BR11	50	None	+	Moderate (10% of pastures)
BR12	16	UASCC (Case No. E24)	-	Moderate (6% of pastures)

/continued...

TABLE 74 /continued...

Farm	No. Animals Examined	Types/Nos. of Malignant Neoplasms in Referred Adult Animals	Incident(s) of Acute Bracken Poisoning	Severity of Bracken Infestation
NBR1	61	None	-	Nil
NBR2	90	None	-	Nil
NBR3	57	None	-	Nil
NBR4	42	None	-	Nil
NBR5	67	None	-	Nil

RESULTS

On the twelve bracken infested farms the hard and soft palates of 608 animals aged 18 months and older were examined for the presence of papillomas. 391 (64.3%) of the animals were born, and had lived their entire lives, on the farm on which they were examined (i.e. they were "home bred") and the remaining 218 (35.7%) were not born on the farm on which they were examined but were purchased and moved onto the farm at a later date (i.e. they were "bought in").

Papillomas were identified in a total of 207 animals (34.0%) and on individual farms the proportion of animals affected ranged between 14.0 and 57.1 percent. 36.8 percent of the home bred animals were found to have papillomas with a range of between 12.5 and 72.7 percent affected on individual farms and 28.9 percent of the bought in animals had papillomas with a range of 0 to 45.5 percent affected on individual farms (Table 75). On eight of the ten farms on which both home bred and bought in animals were present, the proportion of home bred animals with papillomas was greater than that of the bought in animals.

No difference is apparent in the prevalence of palatine papillomas on the farms from which cases of upper alimentary squamous cell carcinoma or malignant urinary bladder neoplasia had been referred (i.e. Farm Nos. BR1, BR2, BR4, BR5, BR7, BR10 and BR12) compared with that on

TABLE 75 The Presence of Palatine Papillomas in Animals on Bracken Infested Farms

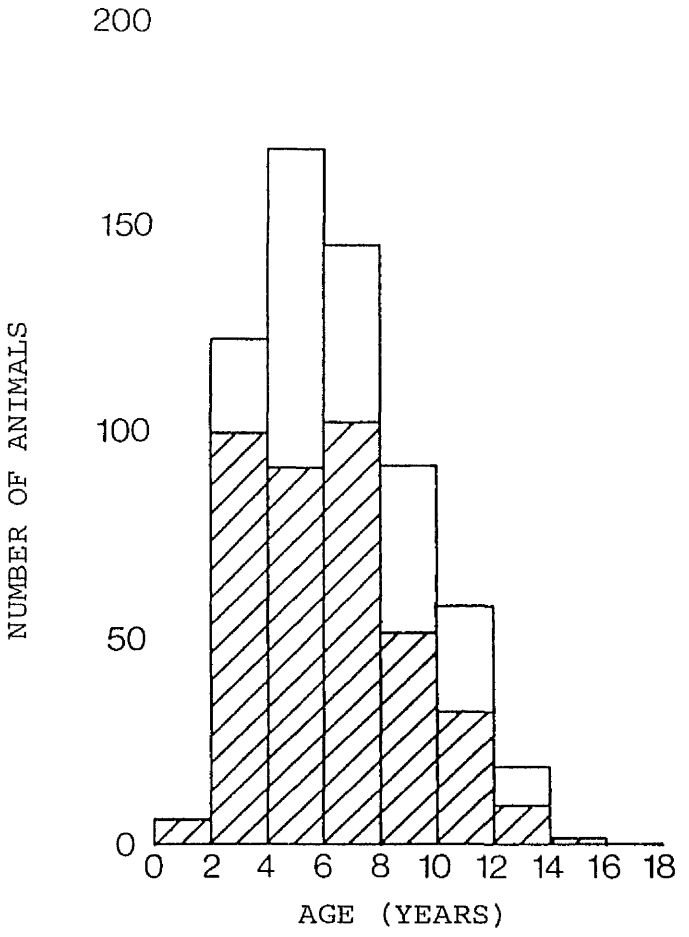
Farm Number	Total Number of Animals Examined	Number of Home Bred Animals	Number of Bought in Animals	Percentage of Animals with Upper Alimentary Papillomas	
				Home Bred	Bought in
BR1	31	11	20	72.7	30.0
BR2	30	22	8	68.2	0.0
BR3	28	17	11	64.7	45.5
BR4	22	12	10	58.3	30.0
BR5	30	12	18	50.0	33.3
BR6	36	17	19	41.2	26.3
BR7	167	167	0	40.3	-
BR8	101	68	33	36.8	18.2
BR9	72	25	47	36.0	42.5
BR10	26	16	10	31.3	40.0
BR11	50	24	26	12.5	15.4
BR12	16	0	16	-	25.0
TOTALS	608	391	218	36.8	28.9

the farms from which such cases had not been referred (i.e. Farm Nos. BR3, BR6, BR8, BR9 and BR11). On the former group of farms 37.1 percent of home bred animals and 28.0 percent of bought in animals had papillomas and on the latter group 36.4 and 29.4 percent respectively of home bred and bought in animals were affected. If only these farms are considered from which no cases of any malignancy had been referred (i.e. Farm Nos. BR3, BR6 and BR11) a very similar situation emerges with 36.2 percent of home bred animals and 25.0 percent of bought in animals affected by palatine papillomas.

On all the bracken infested farms where examinations were performed the severity of infestation was either moderate or severe and acute bracken poisoning incidents had been observed on nine of the twelve farms. No association could be identified between the severity of bracken infestation or the occurrence of acute bracken poisoning and the prevalence of palatine papillomas.

The proportion of animals affected by palatine papillomas in differing age groups is recorded in Figure 55. It is evident that there is a gradual increase with age in the proportion of animals affected by papillomas and a significant correlation ($p = < 0.01$) exists between age and the prevalence of upper alimentary papillomas. When only home bred animals are considered the correlation between age and the prevalence of papillomas is significant ($p = < 0.01$) but this correlation is not maintained when only bought in animals are considered.

(A)



(B)

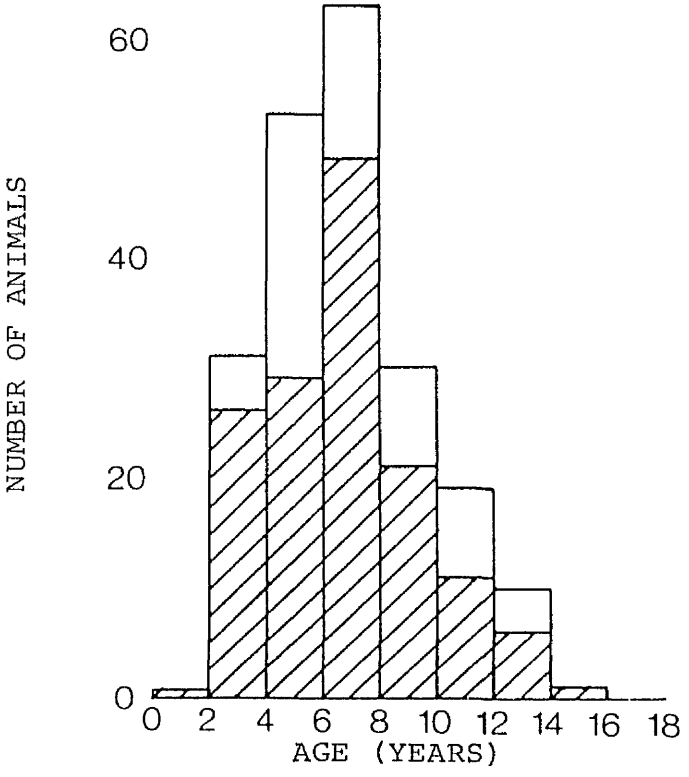


FIGURE 55 The age distribution of animals examined for palatine papillomas (A) and the age distribution of animals with palatine papillomas (B). (Hatched areas indicate home bred animals).

On the five bracken-free farms the hard and soft palates of 316 animals aged 17 months and older were examined for the presence of papillomas. 127 (40.2%) of these animals were home bred and 189 (59.8%) were bought in. Papillomas were identified in a total of 15 animals (4.7%) and on individual farms the proportion of affected animals ranged between 0 and 9.8 percent (Table 76). Only one (0.8%) home bred animal was found in which a palatine papilloma could be identified. The remaining 14 animals with papillomas were bought in, 13 between the ages of one and three years and one as a six month old calf. The previous origins of these animals could not be readily identified but from their ear tags it was evident that their origins were extremely diverse and included farms in Argyll, Dorset, Cornwell, Ireland and South Wales.

TABLE 76 The Presence of Palatine Papillomas in Animals on Bracken - Free Farms

Farm Number	Total Number of Animals Examined	Number of Home Bred Animals	Number of Bought in Animals	Percentage of Animals with Upper Alimentary Papillomas	
				Home Bred	Bought in
NBR1	61	0	61	-	9.8
NBR2	90	6	84	0.0	7.1
NBR3	57	21	36	0.0	5.6
NBR4	42	34	8	0.0	0.0
NBR5	67	67	0	1.5	-
TOTALS	316	127	189	0.8	7.4

DISCUSSION

The results of this study provide further corroborative evidence of the close association between the presence of upper alimentary papillomas in animals aged 15 months and older and exposure to bracken fern. As in the results presented in the previous section, the number of cases in which upper alimentary papillomas were found in animals which had lived their entire lives on farms where there was no evidence of bracken infestation was extremely low. Thus, as has been stated previously, it would seem likely that bracken fern has an important role in the aetiology of upper alimentary papillomas but is not a prerequisite for their development.

The identification of a bovine papillomavirus in upper alimentary papillomas (Jarrett and others, 1978b) raises the question of the respective roles of bovine papillomavirus and bracken fern in the aetiology of upper alimentary papillomas in cattle. Jarrett and others (1978b) have suggested that upper alimentary papillomas are primarily viral in origin and, that their numbers are amplified as a result of exposure to the mutagenic and immunosuppressive properties of bracken fern. This explanation would account for the small numbers of papillomas found in individual animals examined by these authors in their abattoir survey and which were believed to have mainly originated on bracken free farms, compared with the much larger numbers reported by Jarrett and

others (1978a) in animals affected by upper alimentary squamous cell carcinoma which originated on bracken infested farms.

However, an alternative explanation which appears to be equally supported by the epidemiological and virological data available to date is that upper alimentary papillomas have two distinct aetiologies, one viral and the other related to the chemical carcinogen(s) present in bracken fern. The majority of animals (64%) in which Jarrett and others (1978b) identified upper alimentary papillomas in their abattoir survey were less than four years of age. This would accord with a viral aetiology which would be expected to result in lesions being most commonly observed in young animals which have not developed immunity, as is observed with viral induced cutaneous papillomatosis of cattle (Olson, Gordon, Robl and Lee, 1969). Similarly, the relative absence of upper alimentary papillomas in adult cattle from bracken free farms, as found in the present study, could be ascribed to the development of immunity as a consequence of prior exposure to the virus. In contrast, the age prevalence data with regard to the presence of upper alimentary papillomas in animals on bracken infested farms appears to be consistent with prolonged exposure to a carcinogen. If upper alimentary papillomas develop in these animals solely as a result of exposure to the carcinogen(s) in bracken fern this could account for the absence of any reports of the identification of virus in upper alimentary papillomas recovered from cattle over three years of age from bracken infested farms.

GENERAL DISCUSSION AND CONCLUSIONS

The results of the survey of bovine neoplasia described in Chapter 1, and the clinico-pathological study of upper alimentary squamous cell carcinoma, urinary bladder neoplasia and lymphosarcoma described in Chapter 2 have been discussed in detail within these chapters. Thus, this general discussion is confined to an overall apparaisal of the results obtained in the epidemiological study of bovine neoplasia.

In the course of this study, it has been demonstrated that there is a close association between the occurrence of upper alimentary squamous cell carcinoma, upper alimentary papillomas and urinary bladder neoplasia in cattle,and previous exposure of affected animals to bracken fern. The radiomimetic and carcinogenic properties of this plant have been extensively documented and it would seem probable that they are attributable to the same or chemically similar compounds, although their structures have not been elucidated. In cattle, the radiomimetric properties of bracken fern, apparent in the field as acute bracken poisoning, are readily reproduced experimentally, but confirmation of the carcinogenic properties in this species is limited to its effect on the urinary bladder.

Epidemiological evidence, substantiated in the present study, which indicated that bracken fern is implicated in the aetiology of bovine urinary bladder neoplasia prompted experimental investigations to confirm the role of bracken as a carcinogen in cattle. Subsequently, it was demonstrated

that urinary bladder neoplasms can be induced in this species after prolonged feeding of bracken fern (Sofrenovic and others, 1965; Pamukcu and others, 1967; Price and Pamukcu, 1968) and that the types of neoplasms induced are similar to those observed in field cases, including those in the present study. Although it is generally considered that multifactorial aetiologies operate in the majority of cancers, the concept of a single aetiological agent may be valid for bovine urinary bladder neoplasia and in this respect be analogous to certain situations in man. For example, in man, a number of chemical carcinogens, including beta-napthylamine which can cause urinary bladder cancer, and various forms of radioactive materials are believed to induce neoplasia in the absence of other aetiological agents,although, in the individual, a variety of extrinsic and intrinsic factors can mitigate their carcinogenic effects (Weisburger, 1973; Upton 1973).

In contrast, a multifactorial aetiology may be appropriate with regard to upper alimentary squamous cell carcinoma. It would seem highly probable that upper alimentary squamous cell carcinoma results from the malignant transformation of upper alimentary papillomas as described by Jarrett (1978) and thus, initially, it is pertinent to consider the aetiology of upper alimentary papillomas. Two potential aetiological agents have been identified, a papillomavirus (Jarrett and others, 1978b) and, in the present study, bracken fern, but their respective roles in the initiation and

development of upper alimentary papillomas remains to be clarified. Although the induction of upper alimentary papillomas by papillomavirus has been reported (Jarrett, 1978), it has not been ascertained whether all papillomas of the upper alimentary tract are initially virus-associated. Even if it is assumed that all upper alimentary papillomas are virus-associated, it would appear that exposure to bracken fern is an essential component of their development, to account for their high prevalence on bracken infested compared with bracken free farms,as observed in the present study. Possibly, the role of bracken fern could be the activation of latent virus infection as occurs in certain forms of mouse leukaemia in which radiation activates a latent leukaemia virus (Upton and Cosgrove, 1968). However, as discussed earlier, an alternative explanation of the currently available epidemiological and virological data is that upper alimentary papillomas have two distinct aetiologies, and that the aetiology of papillomas which ultimately undergo transformation to carcinoma may be solely related to bracken fern.

Irrespective of the factors involved in the induction of upper alimentary papillomas, exposure to bracken fern would appear to be a pre-requisite for subsequent malignant transformation as the occurrence of upper alimentary squamous cell carcinoma was invariably associated with prior exposure. However it is evident that the rationalisation of any interaction between the potential aetiological agents which have been associated with upper alimentary papillomas and upper alimentary

squamous cell carcinoma will require further investigation before the mechanisms of the initial neoplastic transformation and subsequent malignant transformation can begin to be understood.

During the present study it was not possible to quantify the intake of bracken fern or, more significantly, the carcinogen(s) which it contains, by animals affected by alimentary or urinary bladder neoplasms. Thus,conclusions regarding any dose response relationship are precluded on this basis. However, if the development of both upper alimentary squamous cell carcinoma and urinary bladder neoplasia are purely dose dependant, it would be expected that the neoplasm with the lower dose threshold would invariably be present in animals affected by the neoplasm with the higher dose threshold. In contrast, it was observed that these forms of neoplasia can occur both independently and simultaneously. This would suggest that their development is not a linear function of the dose of carcinogen ingested and that other factors influence the events which take place in the neoplastic process. Such factors may include the age at which ingestion of bracken fern occurs. An age susceptibility to the carcinogen(s) in bracken has been demonstrated in the rat with respect to intestinal adenocarcinoma (Evans and Widdop, 1966; Widdop, 1967),in that rats over one year of age were found to be less susceptible to its carcinogenic effects than those aged six weeks when feeding of bracken fern was initiated. Similarly, if the radiomimetric and carcinogenic properties of bracken fern are synonymous, then an age dependence of tumour induction could exist which is

analagous to that observed in irradiation-induced lymphomas in a variety of species (Duplan, 1976) and suggested for irradiation induced bone tumours in man (Upton, 1973). It is recognised that the induction of neoplasia is a process which depends on a complex interaction of many variables which may include constitutional, environmental and dose factors and that investigation of these variables is a daunting task. However, bracken fern may provide a model for such investigations. In experimental studies designed to investigate the carcinogenicity of bracken fern, it has been demonstrated that intestinal adenocarcinoma can be readily induced in bracken-fed rats whereas, in individual studies, the proportion of animals in which there is simultaneous induction of urinary bladder neoplasia can range between zero and over 80 percent (Pamukcu and Price, 1969; Pamukcu and others, 1970; Schacham and others, 1970; Hirono and others, 1970; Hirono and others, 1973). Detailed examination of the factors which regulate the development of neoplasia in bracken-fed rats may help to provide an explanation of field observations in cattle.

In addition to the association which has been demonstrated in the present study between upper alimentary and urinary bladder neoplasms and exposure to bracken fern, there is some evidence that a similar association exists with regard to intestinal adenocarcinoma of cattle. However, due to the small number of cases in which this latter neoplasm was identified in isolation, an independent statistical assessment of the relationship was not possible. The epidemiology of

intestinal adenocarcinoma in cattle has never been studied previously, the recognition of areas of high frequency of this neoplasm in sheep in New Zealand (Dodd, 1960; Simpson, 1972a), Australia (McDonald and Leaver, 1965; Ross, 1980), Iceland (Georgsson and Vigfusson, 1973) and the U.K. (McCrae and Head, 1978) resulted in investigations to identify the aetiology in this species. Subsequently,a number of factors have been associated with ovine intestinal adenocarcinoma including crested dogstail and potassic fertilisers (Simpson, 1972b) and herring meal containing nitrosamines, particularly dimethylnitrosamine, which, until recently, was used in concentrate feeds for sheep in Iceland (Georgsson and Vigfusson, 1973). Although Simpson (1972b) considered that bracken fern was unlikely to be a causative factor of ovine intestinal adenocarcinoma in New Zealand and Georgsson and Vigfusson (1973) eliminated an aetiological role for bracken fern on the basis that it does not grow in Iceland, the involvement of bracken fern was suspected in the United Kingdom (McCrae and Head, 1981). However, in an experimental study by these authors, prolonged feeding of bracken fern to sheep over periods ranging between 26 and 62 months failed to induce intestinal adenocarcinoma, but resulted in the development of urinary bladder neoplasms in seven of the eight experimental animals, fibrosarcoma of the maxilla and mandible in one animal and a papilloma of the rumen in one animal. At present, it is not known whether a high frequency of intestinal adenocarcinoma in cattle also occurs in areas of high frequency in sheep but, recently,

Johnstone, Alley and Jolly (1983) have suggested that this may be the case in New Zealand and that a similar aetiology may appertain to both species.

It is evident that further epidemiological studies are required to confirm or refute the aetiological role of bracken fern in the development of intestinal adenocarcinoma in either of these species. In addition, the significance of the intestinal adenomas and adenomatous hyperplasia which were observed in many of the cattle with alimentary or urinary bladder neoplasms merits detailed investigation, particularly in light of the strong and consistent association between adenomatous polyps and colonic cancer in man (Correa and Haenszel, 1978).

In conclusion, the data presented in this thesis indicates that a high prevalence of upper alimentary and urinary bladder neoplasms exists in cattle in localised areas of Scotland and that their occurrence is associated with exposure to bracken fern. It is well established that bracken fern contains a potent carcinogen, but it may represent only one factor in the aetiology of these neoplasms and, in the particular case of upper alimentary squamous cell carcinoma, the possible interaction between bracken fern and a papillomavirus remains to be resolved. However, the implication of a plant carcinogen in the aetiology of bovine neoplasia may have considerable importance when the problems of carcinogenesis in man are being considered.

APPENDIX 1

APPENDIX 1.1

Malignant Neoplasms Identified in Animals Examined between 1.9.71 and 31.8.79

Type	Site	Number of Cases
Adenocarcinoma	Intestine	18
	Lung	1
	Urinary Bladder	2
	Uterus	1
Carcinoma	Adrenal Gland	1
	Bronchus	4
	Mammary Gland	1
	Ovary	2
	Thyroid	3
	Uterus	3
Carcinoma (Squamous cell)	Intestine	1
	Ocular	4
	Upper Alimenary Tract	97
	Urinary Bladder	1
	Vagina	1
	Primary Site Not Identified	2
Carcinoma (Transitional cell)	Urinary Bladder	24
Cholangiocarcinoma	Liver	5
Fibrosarcoma	Limb	1
	Mammary Gland	1
	Mandible	3
	Peritoneum	1
	Primary Site Not Identified	1
Granulosa Cell Tumour	Ovary	2
Haemangiosarcoma	Kidney	1
	Skin	2
	Urinary Bladder	5

Type	Site	Number of Cases
Lymphosarcoma	Lymph Nodes	44
	Thymus	33
Maligant Melanoma	Primary Site Not Identified	1
Mesothelioma	Peritoneum	1
Osteosarcoma	Pelvis	1
Sarcoma	Abdominal cavity	1
	Cranial cavity	1
	Spleen	1
	Primary Site Not Identified	1
Teratoma	Ovary	1
	Testis	1
Thecoma	Ovary	1

APPENDIX 1.2

Benign Neoplasms Identified in Animals with Malignant Neoplasia

Type	Site	Number of cases	Case Numbers (Appendix 4)
Adenoma	Gall Bladder	2	E16, E22
	Bile duct	2	E53, E82
	Renal cortex	2	E24, E77
	Thyroid	2	E82, E164
	Pituitary	1	E174
	Adrenal cortex	1	E178
Fibroleiomyoma	Uterus	1	E25
Fibropapilloma	Teat	3	E36, E60, E198
Fibroma	Oesophagus or Rumen	4	E13,E30,E83,E17
Haemangioma	Endometrium	1	E69
	Nasal epithelium	1	E221
	Urethra	1	E12
Lipoma	Colon	2	E14, E20
	Rumen	1	E80
Melanoma	Pelvic connective tissue	1	E77
Papilloma	Perineal skin	1	E35
Phaeochromocytoma	Adrenal gland	4	E15,E19,E25,E42

APPENDIX 2

APPENDIX 2.1

Epidemiological and Pathological Data of Clinical Cases of Upper Alimentary Squamous Cell Carcinoma and Urinary Bladder Neoplasia

CLINICAL CASE NUMBER	SEE APPENDIX 4 CASE NUMBER	CLINICAL CASE NUMBER	SEE APPENDIX 4 CASE NUMBER
A8 -	E9	D1 -	E2
A9 -	E77	D2 -	E71
A10 -	E17	D3 -	E6
A12 -	E21	D4 -	E73
A14 -	E65	D5 -	E79
A15 -	E37	D6 -	E22
A16 -	E39	D7 -	E25
B1 -	E1	D8 -	E30
B3 -	E3	D9 -	E36
B4 -	E70	D10 -	E46
B5 -	E4	D11 -	E48
B6 -	E7	D12 -	E49
B7 -	E11	F1 -	E27
B8 -	E15	F3 -	E45
B9 -	E16	U1 -	E166
B10 -	E20	U2 -	E167
B11 -	E29	U3 -	E168
C1 -	E5	U4 -	E169
C2 -	E8	U5 -	E170
C3 -	E75	U6 -	E190
C4 -	E13	U7 -	E78
C5 -	E14	U8 -	E173
C7 -	E18	U9 -	E186
C8 -	E19	U10 -	E174
C9 -	E24	U11 -	E175
C10 -	E26	U12 -	E191
C11 -	E28	U13 -	E176
C12 -	E32	U14 -	E192
C13 -	E38	U15 -	E193
		U16 -	E196
		U17 -	E177
		U18 -	E82
		U19 -	E197
		U20 -	E178
		U21 -	E198
		U22 -	E180
		U23 -	E181
		U24 -	E200
		U25 -	E201
		U26 -	E182
		U27 -	E183

CLINICAL CASE NUMBER: A1

BREED: SHORTHORN X AGE 9yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx

UAP Tongue, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A2

BREED: SHORTHORN X AGE > 10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, palate, oesophagus

UAP Palate, oesophagus (> 10)

IAC -

MUBN -

BUBN Haemangioma

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A3

BREED: HIGHLAND AGE >10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, palate

UAP Oesophagus, rumen (5)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Ayr Market

CLINICAL CASE NUMBER: A4

BREED: GALLOWAY AGE 8 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue

UAP Palate, oesophagus, rumen (>5)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A5

BREED: ABERDEEN ANGUS X AGE >10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue

UAP Palate, oesophagus (> 10)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Paisley Market

CLINICAL CASE NUMBER: A6

BREED: ABERDEEN ANGUS X AGE > 10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, oesophagus (6)

IAC Duodenum

MUBN -

BUBN -

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A7

BREED: GALLOWAY X AGE > 10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus, oesophageal groove, rumen

UAP Oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A11

BREED: SHORTHORN XAGE >10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus, oesophageal groove, rumen

UAP Palate, oesophageal groove (> 5)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: A13

BREED: ABERDEEN ANGUS AGE > 8 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx, oesophagus

UAP Palate, oesophagus,

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of the small intestine

ORIGINS Dalmally Market

CLINICAL CASE NUMBER: B2

BREED: HIGHLAND AGE > 10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER -

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: C6

BREED: ABERDEEN ANGUS AGE > 8 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia

UAP Tongue, palate (3)

IAC -

MUBN -

BUBN Haemangioma

OTHER Adenomatous hyperplasia of the small intestine

ORIGINS Cattle dealer

CLINICAL CASE NUMBER: F2

BREED: ABERDEEN ANGUS AGE > 10 yrs SEX FEMALE

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate (2)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum and colon

ORIGINS Cattle dealer

APPENDIX 3

APPENDIX 3.1

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA (i) OROPHARYNGEAL SYNDROME

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
A1	33.5	10.8	7.52	45	32.2	15.1	43	56	1	0
A2	25.5	-	5.30	48	-	12.2	50	49	1	0
A3	23	7.8	-	-	33.9	6.8	45	54	1	0
A4	22.5	7.4	4.77	47	32.9	9.1	65	35	0	0
A5	24.5	8.4	4.21	58	34.3	9.2	57	36	7	0
A6	40	-	-	-	-	15.5	57	43	0	0
A7	32	9.3	6.13	52	29.0	9.2	66	34	0	0
A8	34	11.0	6.36	54	32.4	8.5	71	28	1	0
A9	26	8.8	3.46	75	33.9	6.6	43	56	1	0
A10	32	10.8	5.13	62	33.8	6.5	57	43	0	0
A11	25	7.6	5.48	46	30.4	5.1	19	73	8	0
A12	27.5	9.4	5.20	53	34.2	15.6	43	57	0	0
A13	25	9.1	4.12	61	36.4	7.1	42	54	4	0
A14	29.5	8.8	5.65	52	29.8	7.1	71	27	2	0
A15	28	9.5	4.64	60	34.0	8.6	42	58	0	0
A16	28.5	9.1	4.72	60	32.0	2.5	18	82	0	0

APPENDIX 3.2

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(i) OROPHARYNGEAL SYNDROME

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (μmol/l)	ALKALINE PHOS-PHATASE (IU/l)	ASPARTATE AMINO-TRANS-FERASE (IU/l)	ALANINE AMINO-TRANS-FERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
A1	3.0	142	4.2	96	2.15	0.95	1.26	-	-	115	25	73	13	60
A2	5.6	136	3.2	97	-	-	1.55	5	43	147	25	84	20	64
A3	7.6	136	4.0	-	2.48	0.53	2.03	5	28	55	19	87	15	72
A4	4.6	139	4.6	94	-	-	1.93	3	14	57	14	58	10	48
A5	5.0	-	-	-	-	-	1.22	3	21	133	7	78	16	62
A6	3.7	138	2.8	92	-	-	1.94	3	70	161	30	83	22	61
A7	6.0	145	5.3	89	2.45	0.49	1.55	10	42	54	20	96	22	74
A8	35.9	122	4.0	91	2.83	1.27	2.32	5	113	101	24	94	21	73
A9	1.7	137	5.4	89	2.65	0.53	1.49	2	42	93	29	92	19	73
A10	2.0	135	5.0	97	2.65	0.70	1.94	2	42	252	24	95	24	71
A11	6.3	133	3.7	100	2.18	0.49	1.71	2	28	49	42	71	13	58
A12	4.8	143	7.4	86	1.85	0.16	1.97	7	57	229	39	100	21	79
A13	2.5	139	3.7	107	3.00	0.37	2.07	7	21	139	15	82	22	60
A14	2.5	141	4.7	98	2.60	0.50	1.95	1	72	79	26	103	22	81
A15	4.3	133	5.0	112	2.30	0.71	0.50	4	30	88	34	95	27	68
A16	3.6	133	3.0	100	2.08	0.40	1.65	10	35	309	22	94	16	78

APPENDIX 3.3

URINE AND FAECAL EXAMINATIONS AND SERUM PEPSINOGEN ESTIMATIONS IN ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(i) OROPHARYNGEAL SYNDROME

CASE NUMBER	URINE		FAECES			SERUM PEPSINOGEN (MU Tyrosine)
	Protein (mg/100mls)	Erythrocytes	Trichostrongyle Eggs (Eggs per gram)	Acid fast Bacilli Resembling *Mycobacterium Paratuberculosis*		
				Initial Sample	Repeat Sample(s)	
A1	35	0	0	Negative	Negative	-
A2	51	+++	50	Negative	-	2560
A3	6	0	-	-	-	-
A4	23	0	0	Negative	Negative(2)	-
A5	-	-	-	-	-	-
A6	0	0	-	-	-	-
A7	29	0	200	Inconclusive	Negative(2)	1528
A8	166	0	-	-	-	-
A9	86	+	-	-	-	-
A10	7	0	-	-	-	-
A11	0	0	-	-	-	-
A12	73	+++	5800	Inconclusive	Negative	2072
A13	9	0	0	Inconclusive	Negative(3)	1999

APPENDIX 3.3 (cont'd)

CASE NUMBER	URINE		FAECES			SERUM PEPSINOGEN (MU Tyrosine)
	Protein (mg/100mls)	Erythrocytes	Trichostrongyle Eggs (Eggs per gram)	Acid fast Bacilli Resembling *Mycobacterium Paratuberculosis*		
				Initial Sample	Repeat Sample(s)	
A14	7	0	-	-	-	-
A15	21	0	-	-	-	-
A16	121	0	200	Negative	-	776

APPENDIX 3.4

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(ii) OESOPHAGEAL SYNDROME

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
B1	15.5	5.4	2.25	69	34.8	1.1	19	80	1	0
B2	25.0	-	-	-	-	5.0	-	-	-	-
B3	33.5	-	-	-	-	4.0	53	46	1	0
B4	23	7.3	4.28	54	31.7	18.7	81	18	1	0
B5	36	12.3	6.61	55	34.2	3.5	56	43	1	0
B6	21.5	6.6	3.74	58	30.7	4.8	45	50	5	0
B7	27	8.5	5.01	54	31.5	6.7	58	42	0	0
B8	35	10.7	5.96	59	30.6	6.7	65	34	1	0
B9	38	11.9	6.74	56	31.3	9.2	61	37	2	0
B10	19.5	6.3	3.81	51	32.3	10.5	69	26	5	0
B11	31	10.7	5.63	55	34.5	8.5	46	50	4	0

APPENDIX 3.5

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(ii) OESOPHAGEAL SYNDROME

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (µmol/l)	ALKALINE PHOS-PHATASE (IU/l)	ASPARTATE AMINO-TRANS-FERASE (IU/l)	ALANINE AMINO-TRANS-FERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
B1	3.7	139	3.4	101	2.05	0.21	1.13	10	28	350	20	82	17	65
B2	4.5	137	3.6	96	2.38	0.74	1.68	2	28	89	15	75	20	55
B3	3.2	136	4.1	95	2.20	0.49	1.13	3	142	214	16	72	12	60
B4	2.8	134	4.7	100	2.05	0.25	1.62	5	114	127	28	86	14	72
B5	4.2	142	4.4	106	2.43	0.86	2.49	22	50	120	10	77	35	42
B6	1.5	132	3.2	104	2.13	0.74	0.68	10	14	71	12	76	15	61
B7	6.1	141	4.7	109	2.33	0.70	0.87	5	43	300	34	79	19	60
B8	4.2	135	3.9	95	2.15	0.70	1.78	15	85	272	32	85	19	66
B9	4.8	149	4.2	95	2.48	0.70	1.39	2	43	61	27	82	23	59
B10	4.8	140	3.8	100	2.35	0.66	1.32	3	78	62	42	90	15	75
B11	7.6	135	4.3	104	2.40	0.82	1.45	9	43	116	41	99	31	68

APPENDIX 3.6

URINE AND FAECAL EXAMINATIONS AND SERUM PEPSINOGEN ESTIMATIONS IN ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(ii) OESOPHAGEAL SYNDROME

CASE NUMBER	URINE		FAECES			SERUM PEPSINOGEN (MU Tyrosine)
	Protein (mg/100mls)	Erythrocytes	Trichostrongyle Eggs (Eggs per gram)	Acid fast Bacilli Resembling *Mycobacterium Paratuberculosis*		
				Initial Sample	Repeat Sample(s)	
B1	0	0	50	Negative	-	972
B2	3	0	-	-	-	-
B3	5	0	300	Negative	-	1862
B4	36	0	50	Negative	Negative	844
B5	53	0	0	Negative	-	1566
B6	0	0	100	Negative	-	1274
B7	41	0	150	Negative	Negative	1810
B8	14	0	-	-	-	-
B9	0	0	-	-	-	-
B10	18	0	0	Negative	Negative	1622

APPENDIX 3.7

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA (iii) RUMENAL TYMPANY SYNDROME

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
C1	23.5	6.7	4.19	56	29.4	4.9	53	47	0	0
C2	32	8.8	6.46	50	27.5	10.4	75	25	0	0
C3	24	6.9	4.86	49	28.8	9.9	64	35	1	0
C4	34	11.6	5.75	59	34.1	5.2	40	57	3	0
C5	35	12.9	6.66	53	36.9	6.6	56	44	0	0
C6	25	8.5	4.29	58	34.0	3.2	34	64	2	0
C7	26	8.8	4.27	61	33.9	3.2	21	79	0	0
C8	27	8.5	5.20	52	31.5	8.2	60	39	1	0
C9	26	7.2	4.62	56	27.8	9.8	77	21	1	1
C10	34.5	11.5	4.69	74	33.3	4.2	68	31	1	0
C11	35	11.0	6.84	51	31.4	7.0	32	54	14	0
C12	33	11.3	5.69	58	34.2	6.0	39	56	3	2
C13	20	6.9	3.67	54	34.5	5.2	38	62	0	0

APPENDIX 3.8

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(iii) RUMENAL TYMPANY SYNDROME

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (µmol/l)	ALKALINE PHOS-PHATASE (IU/l)	ASPARTATE AMINO-TRANS-FERASE (IU/l)	ALANINE AMINO-TRANS-FERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
C1	6.0	140	4.0	100	-	-	1.90	5	57	11	17	98	10	88
C2	4.6	132	3.6	105	2.32	0.66	1.16	24	135	55	8	96	16	80
C3	7.1	130	3.5	100	2.08	0.45	1.10	3	114	350	43	77	17	60
C4	5.0	141	4.9	103	2.13	0.58	1.97	2	50	73	67	71	12	59
C5	7.1	140	4.9	98	2.25	0.37	2.52	10	106	273	55	82	23	59
C6	4.3	138	4.2	100	2.03	0.37	2.16	2	78	153	10	87	15	72
C7	2.7	152	3.9	109	2.53	0.53	2.16	5	28	109	48	80	19	61
C8	8.0	146	4.3	101	1.88	0.58	2.65	9	57	153	29	86	21	65
C9	12.8	141	3.7	81	2.15	0.58	2.94	2	50	105	44	97	16	81
C10	15.3	131	3.2	101	2.33	0.37	1.23	7	57	96	19	98	22	76
C11	12.8	144	4.1	98	2.63	0.58	2.97	3	163	247	42	101	24	77
C12	8.1	153	5.6	100	2.38	0.53	1.58	1	43	124	41	88	22	66
C13	5.1	140	3.8	99	1.90	0.62	1.98	11	34	118	28	72	15	57

APPENDIX 3.9

URINE AND FAECAL EXAMINATIONS AND SERUM PEPSINOGEN ESTIMATIONS IN ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA (iii) RUMENAL TYMPANY SYNDROME

CASE NUMBER	URINE		FAECES			SERUM PEPSINOGEN (MU Tyrosine)
	Protein (mg/100mls)	Erythrocytes	Trichostrongyle Eggs (Eggs per gram)	Acid fast Bacilli Resembling Mycobacterium Paratuberculosis		
				Initial Sample	Repeat Sample(s)	
C1	9	0	-	-	-	-
C2	65	0	-	-	-	-
C3	225	+++	50	Negative	-	1509
C4	43	0	150	Negative	Negative	
C5	66	++	-	-	-	-
C6	4	0	0	Negative	-	1280
C7	8	0	0	Negative	-	1308
C8	36	0	400	Negative	-	1451
C9	3	0	150	Negative	-	1260
C10	26	0	-	-	-	-
C11	32	0	0	Negative	-	1992
C12	42	0	0	Negative	-	1494
C13	90	++	50	Negative	-	589

APPENDIX 3.10

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA (iv) WASTING AND DIARRHOEA SYNDROME

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
D1	23.5	7.9	4.32	54	33.6	15.7	81	19	0	0
D2	14.0	3.8	2.50	56	27.1	6.3	76	24	0	0
D3	27	7.5	5.31	51	27.8	4.0	61	39	0	0
D4	23	7.2	4.06	57	31.3	5.6	45	50	5	0
D5	18.5	6.0	3.93	46	32.4	3.6	43	55	2	0
D6	27.5	9.4	4.41	62	34.2	6.1	5	94	1	0
D7	26	8.7	4.61	56	33.5	8.7	49	41	8	0
D8	35	11.6	6.58	53	33.1	8.5	69	30	1	0
D9	22.5	7.8	3.54	64	34.6	5.8	40	60	0	0
D10	27	7.9	4.36	62	29.3	7.1	53	42	5	0
D11	32	11.3	5.70	56	35.3	4.4	50	48	2	0
D12	24	7.0	5.15	47	29.2	12.1	59	40	1	0

APPENDIX 3.11

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(iv) WASTING AND DIARRHOEA SYNDROME

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (µmol/l)	ALKALINE PHOS-PHATASE (IU/l)	ASPARTATE AMINO-TRANS-FERASE (IU/l)	ALANINE AMINO-TRANS-FERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
D1	10.5	131	1.6	100	2.00	0.66	0.90	7	121	94	16	78	14	64
D2	6.0	135	4.7	100	2.00	0.45	1.16	9	28	-	-	66	11	55
D3	4.2	134	4.6	94	2.35	0.53	1.36	15	57	174	20	88	13	75
D4	4.3	141	3.9	104	2.63	0.86	1.29	7	78	77	34	85	17	68
D5	3.5	143	4.6	105	2.30	0.58	1.68	2	21	89	17	69	13	56
D6	6.8	135	4.4	103	2.33	0.58	1.16	2	36	89	25	91	24	67
D7	3.8	154	4.3	110	2.33	0.53	1.94	2	85	126	40	88	16	72
D8	6.0	138	3.4	106	2.73	0.70	1.45	2	64	118	43	94	22	72
D9	1.8	137	4.5	110	2.14	0.38	1.17	6	57	125	16	93	9	84
D10	5.8	150	2.0	97	2.13	0.30	1.38	1	123	221	18	78	21	57
D11	6.2	143	3.3	91	2.22	0.86	0.93	1	23	114	47	93	30	63
D12	5.5	138	3.8	99	3.59	0.33	1.09	3	60	359	90	87	13	74

APPENDIX 3.12

URINE AND FAECAL EXAMINATIONS AND SERUM PEPSINOGEN ESTIMATIONS IN ANIMALS AFFECTED BY UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA

(iv) WASTING AND DIARRHOEA SYNDROME

CASE NUMBER	URINE		FAECES			SERUM PEPSINOGEN (MU Tyrosine)
	Protein (mg/100mls)	Erythrocytes	Trichostrongyle Eggs (Eggs per gram)	Acid fast Bacilli Resembling Mycobacterium Paratuberculosis		
				Initial Sample	Repeat Sample(s)	
D1	27	O	50	Negative	Negative	1010
D2	85	++	O	Negative	Negative	650
D3	93	O	1150	Negative	Negative	872
D4	O	O	O	Negative	Negative(2)	1352
D5	48	O	200	Negative	Negative	1248
D6	83	O	O	Positive	Inconclusive Negative	2714
D7	O	O	O	Negative	Negative	1381
D8	88	O	O	Negative	Negative	1920
D9	87	O	O	Negative	Negative	612
D10	5	O	O	Negative	Negative(3)	2430
D11	O	O	O	Negative	Negative	1875
D12	62	O	O	Negative	Negative	1159

APPENDIX 3.13

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY URINARY BLADDER NEOPLASIA

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($\times 10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($\times 10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
U1	22	7.3.	3.43	64	33.2	17.8	55	45	0	0
U2	30	10.7	5.34	56	35.7	7.1	54	45	1	0
U3	10	3.0	1.42	70	30.0	5.8	37	60	3	0
U4	35	11.0	6.71	52	31.4	11.0	28	68	4	0
U5	38.5	12.3	8.34	46	31.9	8.3	-	-	-	-
U6	39	-	-	-	-	13.8	-	-	-	-
U7	30.5	10.5	5.12	60	34.4	4.7	32	55	13	0
U8	21.5	6.6	2.68	80	30.7	5.0	63	37	0	0
U9	19	5.6	3.10	61	29.5	5.1	67	31	0	2
U10	21	6.3	3.42	61	30.0	7.6	51	47	2	0
U11	25	8.2	5.25	48	32.8	6.3	40	58	2	0
U12	33	10.4	5.99	55	31.5	8.0	45	45	8	2
U13	15.5	5.3	2.81	55	34.2	5.1	35	63	1	1
U14	12.5	3.8	1.90	66	30.4	6.2	42	51	0	7
U15	22.5	7.5	3.84	59	33.3	4.1	41	56	3	0
U16	20	6.9	3.24	62	34.5	3.7	47	53	0	0
U17	32.5	11.0	5.24	62	33.9	7.9	44	56	0	0
U18	23	7.8	3.54	65	33.9	4.7	49	44	7	0
U19	29	9.5	5.37	54	32.8	5.2	44	53	3	0
U20	28	9.1	4.67	60	32.5	5.5	60	39	1	0
U21	29	10.7	6.02	48	36.8	4.1	14	76	10	0
U22	14	3.8	1.87	75	27.1	5.8	53	43	4	0
U23	30	10.4	5.23	57	34.7	4.9	52	48	0	0
U24	8	1.9	1.05	76	23.8	12.5	59	38	0	3
U25	38	12.0	6.5	58	31.6	9.4	48	34	18	0
U26	8	1.5	0.71	113	18.7	2.6	48	52	0	0
U27	23	7.9	3.92	59	34.3	5.9	57	41	0	2

APPENDIX 3.14

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY URINARY BLADDER NEOPLASIA

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (μmol/l)	ALKALINE PHOSPHATASE (IU/l)	ASPARTATE AMINOTRANSFERASE (IU/l)	ALANINE AMINOTRANSFERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
U1	13.4	135	3.6	86	2.50	1.48	1.26	5	43	42	16	102	19	83
U2	4.8	142	2.8	98	-	0.99	1.32	2	14	74	26	79	30	49
U3	5.1	140	4.9	102	2.35	0.95	3.00	7	71	95	51	64	15	49
U4	4.5	138	3.9	98	2.33	0.82	0.97	7	36	78	30	97	34	63
U5	4.8	139	3.9	95	2.25	0.90	0.90	3	21	90	45	92	37	55
U6	6.0	148	5.2	97	-	0.74	2.23	-	57	173	49	99	32	67
U7	1.3	137	4.1	104	2.35	0.78	1.52	2	57	61	25	79	24	55
U8	7.3	139	3.2	111	3.13	0.99	2.45	5	185	52	13	65	31	34
U9	2.7	141	3.7	105	2.40	0.74	1.03	3	36	161	41	79	19	59
U10	4.0	144	4.6	112	2.30	0.78	1.55	5	14	64	40	68	17	51
U11	65.0	140	4.3	80	2.20	1.24	0.90	3	49	76	25	73	21	52
U12	5.1	143	4.2	96	2.38	0.86	2.29	2	36	71	46	89	27	62
U13	10.1	111	8.2	78	2.25	0.65	3.55	9	50	102	47	57	22	35
U14	7.3	152	4.8	107	2.13	0.82	3.10	3	78	800	68	60	19	41
U15	2.5	136	3.8	106	2.43	0.58	1.07	2	28	123	18	79	23	56
U16	3.8	145	4.1	111	2.00	0.90	1.55	1	28	58	20	78	30	48
U17	3.7	142	3.8	96	2.60	0.66	1.45	1	30	80	41	89	18	71
U18	4.8	150	4.2	108	2.40	0.58	2.22	2	81	133	55	72	24	48
U19	1.6	138	4.2	96	2.54	0.50	1.46	4	50	67	35	91	23	68
U20	2.8	141	3.8	113	2.58	0.70	1.25	2	27	111	50	77	26	51
U21	2.9	150	4.8	103	2.70	0.51	2.05	7	58	96	39	88	33	55
U22	2.9	154	4.9	108	1.66	0.76	1.53	2	188	129	313	52	18	34
U23	8.8	145	4.1	107	2.16	0.57	1.98	6	42	90	8	76	21	55
U24	6.6	132	4.1	90	1.90	0.63	2.85	15	111	195	32	37	9	28
U25	5.3	142	3.6	97	2.38	0.47	2.95	1	184	293	26	81	25	56
U26	8.5	136	5.4	100	1.35	1.02	2.35	3	64	217	86	37	13	24
U27	16.1	139	7.5	89	2.09	0.53	1.56	1	58	110	27	92	20	72

APPENDIX 3.15

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY MULTICENTRIC LYMPHOSARCOMA

CASE NO.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
M1	28	7.9	6.04	46	28.2	9.1	22	62	0	16
M2	33	8.8	6.07	54	26.7	21.9	20	78	0	2
M3	24	7.5	5.54	43	31.3	42.4	2	92	6	0
M4	45	12.2	8.58	52	27.1	7.7	73	20	6	1
M5	29	8.8	4.79	61	30.3	3.7	25	74	0	1
M6	24	6.9	5.71	42	28.8	4.2	56	44	0	0
M7	23	-	-	-	-	14.6	8	92	0	0
M8	22	7.2	6.41	34	32.7	23.4	2	98	0	0
M9	22	6.3	5.12	43	28.6	18.2	22	78	0	0
M10	27	8.2	5.36	50	30.4	10.3	18	82	0	0
M11	14	4.7	2.96	47	33.6	10.3	43	57	0	0
M12	27	8.5	6.74	40	31.5	108.0	1	99	0	0
M13	20	6.5	4.56	44	32.5	3.0	22	77	1	0
M14	22	6.9	4.99	44	31.4	7.9	20	80	0	0
M15	31	8.8	7.62	41	28.4	9.0	18	82	0	0
M16	27.5	9.1	7.68	36	33.1	19.7	43	57	0	0
M17	20	7.0	5.31	38	35.0	114.0	2	0	0	98
M18	27	9.1	6.69	40	33.7	10.8	58	42	0	0
M19	32	-	-	-	-	5.0	18	82	0	0
M20	25	9.5	5.05	50	38.0	10.5	66	34	0	0
M21	27	7.9	6.23	43	29.3	39.5	32	67	1	0
M22	27	9.1	4.54	59	33.7	106.9	4	96	0	0
M23	30	9.8	5.08	59	32.7	104.0	13	87	0	0
M24	23	8.8	6.06	38	38.2	7.1	40	60	0	0
M25	37.5	12.9	8.17	46	34.4	9.1	51	41	8	0
M26	14.5	4.4	2.79	52	30.3	6.1	59	40	0	1
M27	23	-	-		-	7.0	44	56	0	0
M28	25	6.9	3.36	74	27.6	10.9	40	60	0	0

APPENDIX 3.16

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY MULTICENTRIC LYMPHOSARCOMA

CASE NO.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (μmol/l)	ALKALINE PHOSPHATASE (IU/l)	ASPARTATE AMINO-TRANSFERASE (IU/l)	ALANINE AMINO-TRANSFERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
M1	13.1	151	4.6	105	2.80	0.70	3.28	8	45	41	12	80	31	49
M2	13.1	139	4.6	105	1.76	0.82	4.37	14	37	12	9	69	26	43
M3	6.5	139	4.3	115	2.08	0.17	2.10	5	57	51	31	63	29	34
M4	2.9	145	5.5	90	3.00	0.86	2.84	10	258	4	12	75	26	49
M5	12.3	142	4.3	111	2.63	1.03	3.49	9	135	31	29	60	35	25
M6	7.3	133	3.0	116	1.48	0.49	1.40	4	44	94	17	56	26	30
M7	10.5	142	6.0	98	3.03	0.91	4.78	2	28	50	90	56	26	30
M8	22.7	140	4.2	103	3.04	0.66	3.75	22	249	185	52	62	29	33
M9	6.0	144	4.7	100	2.46	0.62	1.73	8	94	99	25	61	27	34
M10	4.0	138	4.6	96	2.55	0.74	1.84	10	43	116	49	84	19	65
M11	18.5	145	5.4	119	1.82	0.98	3.30	10	158	343	52	49	15	34
M12	10.2	147	6.5	106	2.65	1.26	2.81	3	50	-	18	66	28	38
M13	8.6	125	4.1	101	-	-	2.55	17	57	55	23	56	23	33
M14	9.9	144	4.1	100	2.50	0.73	2.92	16	52	125	18	61	26	35
M15	13.0	157	4.1	108	2.70	0.96	3.40	7	81	96	29	65	27	38
M16	6.5	142	4.6	108	2.58	0.78	2.78	12	43	75	33	75	35	40
M17	7.0	146	6.8	99	2.45	1.03	3.46	5	99	121	11	76	25	51
M18	1.5	142	4.0	104	2.45	0.65	2.20	2	171	69	21	80	23	57
M19	4.3	136	4.8	97	2.25	0.98	1.68	5	114	99	19	64	29	35
M20	4.6	146	6.0	100	2.22	0.50	2.96	21	38	150	5	80	20	60
M21	6.7	130	4.2	100	2.29	0.74	3.32	13	141	885	43	86	18	68
M22	7.1	139	4.7	90	2.10	0.56	4.15	28	67	111	20	82	36	46
M23	3.0	141	3.5	94	2.28	0.64	1.78	5	36	118	18	87	21	66
M24	5.4	146	5.9	110	2.10	0.45	1.84	5	42	106	13	72	18	54
M25	4.0	142	3.8	96	2.42	0.66	1.39	1	50	93	8	79	27	52
M26	17.4	141	4.8	94	1.98	1.03	3.00	10	50	297	50	70	25	45
M27	7.0	138	4.4	97	2.08	0.74	2.17	9	50	82	16	78	21	57
M28	3.7	149	4.4	112	1.78	0.66	1.34	1	60	166	24	59	14	45

APPENDIX 3.17

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY THYMIC LYMPHOSARCOMA

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
T1	40	12.0	10.52	38	30.0	7.5	50	49	1	0
T2	33	-	-	-	-	12.9	53	47	0	0
T3	38	12.9	9.08	42	33.9	13.0	37	62	1	0
T4	30	-	-	-	-	7.8	36	64	0	0
T5	24	6.9	4.87	49	28.8	45.9	6	94	0	0
T6	43.5	-	-	-	-	8.3	54	46	0	0
T7	28	8.8	7.91	35	31.4	23.0	19	81	0	0
T8	27	-	-	-	-	6.8	20	80	0	0
T9	25	7.5	6.30	40	30.0	68.1	2	97	0	1
T10	18	5.1	3.05	59	28.3	6.9	31	68	0	1
T11	30	10.9	7.70	39	36.3	6.1	75	25	0	0
T12	29.5	9.9	7.55	39	33.6	18.5	72	27	1	0
T13	29	8.8	6.94	42	30.3	20.8	9	91	0	0
T14	38	14.8	9.71	39	38.9	13.0	47	53	0	0
T15	29	-	-	-	-	8.0	24	76	0	0
T16	36	-	-	-	-	19.4	40	60	0	0
T17	28.5	8.5	5.53	52	29.8	6.8	28	72	0	0
T18	30	9.4	5.74	52	31.3	15.5	21	78	0	1
T19	30	8.8	5.83	51	29.3	6.0	21	79	0	0
T20	31	10.4	5.94	52	33.5	23.0	12	86	0	2
T21	28	7.5	6.35	44	26.8	8.5	61	35	1	0
T22	32.5	11.3	6.46	50	34.8	8.0	34	66	0	0
T23	38	12.3	7.33	52	32.4	11.1	57	43	0	0
T24	24.5	8.8	6.09	40	35.9	7.7	32	63	5	0
T25	32	10.4	5.63	57	32.5	12.9	32	68	0	0
T26	40.5	11.9	7.77	52	29.4	14.8	40	59	1	0
T27	25	8.5	6.36	39	34.0	8.7	46	54	0	0
T28	31	9.1	5.89	53	29.4	5.3	27	73	0	0
T29	32.5	12.4	6.61	49	38.1	7.8	31	67	2	0
T30	30	10.0	6.59	46	33.3	5.3	18	82	0	0

APPENDIX 3.18

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY THYMIC LYMPHOSARCOMA

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (umol/l)	ALKALINE PHOSPHATASE (IU/l)	ASPARTATE AMINO-TRANSFERASE (IU/l)	ALANINE AMINO-TRANSFERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
T1	2.7	140	3.5	102	2.60	0.77	2.76	8	116	77	29	79	26	53
T2	3.6	150	5.7	106	2.79	0.36	2.76	10	54	88	5	68	21	47
T3	10.5	143	4.1	100	2.28	0.74	1.97	2	85	256	63	104	27	77
T4	3.8	150	4.0	98	2.38	0.49	2.42	9	64	164	58	91	30	61
T5	9.7	149	5.4	101	2.22	0.62	2.76	16	49	110	40	80	26	54
T6	3.0	148	4.8	97	2.40	0.78	2.39	9	85	60	30	67	26	41
T7	10.8	134	4.4	95	2.05	0.82	3.07	21	43	300	28	59	21	38
T8	9.1	143	5.1	98	2.60	0.70	3.17	9	28	90	5	66	21	45
T9	8.8	140	4.3	109	2.42	0.84	2.89	8	58	70	28	76	24	52
T10	7.1	147	5.2	105	2.65	0.91	1.87	31	64	122	23	89	29	60
T11	5.0	132	4.0	103	2.54	0.90	2.31	4	107	82	30	74	18	56
T12	2.8	143	4.3	101	2.13	0.62	1.84	5	50	71	15	92	24	68
T13	7.0	132	4.8	89	2.25	0.72	2.66	21	50	245	73	94	29	65
T14	4.5	148	3.8	96	-	-	2.68	9	99	177	73	73	36	37
T15	4.2	149	5.5	96	2.33	2.06	2.78	2	50	94	21	93	22	71
T16	6.6	135	3.7	95	2.20	0.45	1.87	21	64	150	24	85	26	59
T17	2.9	148	3.9	92	2.63	0.78	1.91	8	76	177	45	84	34	50
T18	8.2	134	3.5	103	2.62	0.80	2.54	10	61	162	28	86	22	64
T19	5.8	145	4.1	104	2.25	0.61	1.86	5	80	112	12	82	31	51
T20	4.8	149	4.9	104	2.51	0.63	2.01	1	46	207	29	74	27	47
T21	11.1	143	3.7	94	2.20	0.60	2.69	11	50	509	22	56	20	36
T22	-	142	4.4	98	-	-	1.71	-	64	180	53	85	36	49
T23	8.0	132	3.2	92	2.31	0.55	2.80	22	106	487	88	89	13	67
T24	8.0	138	4.5	96	2.23	0.33	2.84	15	21	120	15	77	27	50
T25	2.9	148	4.5	98	2.35	0.78	2.00	11	57	75	22	67	22	45
T26	2.2	145	4.0	102	2.50	0.78	2.17	2	43	49	26	70	27	43
T27	31.7	120	3.7	67	2.20	1.07	5.10	15	50	183	39	82	18	64
T28	5.0	140	3.8	99	2.40	0.72	2.20	9	30	133	31	89	30	59
T29	6.6	136	4.4	93	2.33	0.62	2.04	15	21	193	15	73	32	41
T30	9.2	152	4.1	94	1.93	0.46	1.87	10	50	34	31	84	26	58

APPENDIX 3.19

HAEMATOLOGICAL PARAMETERS OF ANIMALS AFFECTED BY SKIN LYMPHOSARCOMA

CASE No.	PACKED CELL VOLUME (%)	HAEMOGLOBIN CONCENTRATION (g/100ml)	ERYTHROCYTE COUNT ($x10^6$/ml)	MEAN CELL VOLUME (μ^3)	MEAN CELL HAEMOGLOBIN CONCENTRATION (g/dl)	LEUKOCYTE COUNT ($x10^3$/ml)	NEUTROPHILS (%)	LYMPHOCYTES (%)	EOSINOPHILS (%)	MONOCYTES (%)
S1	29.5	9.9	7.60	39	33.6	9.9	22	73	3	2
S2	24	6.7	5.71	42	27.9	12.5	51	47	2	0
S3	19	6.0	3.55	54	31.5	14.3	26	74	0	0

APPENDIX 3.20

BIOCHEMICAL PARAMETERS OF ANIMALS AFFECTED BY SKIN LYMPHOSARCOMA

CASE No.	UREA (mmol/l)	SODIUM (mmol/l)	POTASSIUM (mmol/l)	CHLORIDE (mmol/l)	CALCIUM (mmol/l)	MAGNESIUM (mmol/l)	INORGANIC PHOSPHATE (mmol/l)	BILIRUBIN (µmol/l)	ALKALINE PHOS-PHATASE (IU/l)	ASPARTATE AMINO-TRANS-FERASE (IU/l)	ALANINE AMINO-TRANS-FERASE (IU/l)	TOTAL PROTEIN (g/l)	ALBUMIN (g/l)	GLOBULIN (g/l)
S1	2.7	138	3.3	104	2.45	0.74	2.39	5	36	59	33	74	23	51
S2	1.9	133	3.6	96	2.56	0.61	1.30	7	12	191	38	77	19	58
S3	9.2	140	4.7	90	2.39	0.89	3.22	30	43	856	61	73	22	51

APPENDIX 4

Abbreviations

UASCC	Upper alimentary squamous cell carcinoma
UAP	Upper alimentary papillomas
IAC	Intestinal adenocarcinoma
MUBN	Malignant urinary bladder neoplasia
BUBN	Benign urinary bladder neoplasia
V.S.	Practising Veterinary Surgeon

APPENDIX 4.1

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA OF ANIMALS WITH UPPER ALIMENTARY SQUAMOUS CELL CARCINOMA.

EPIDEMIOLOGY CASE NUMBER: E1 CLINICAL CASE NUMBER: B1

BREED: Galloway AGE 12 yrs SEX:Female DATE OF REFERRAL: 13.9.71

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia

UAP Oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 1 month of age

BRACKEN INFESTATION Moderate (13% of pasture)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s. and self)

PREVIOUS ORIGINS Purchased from cattle dealer

EPIDEMIOLOGY CASE NUMBER: E2 CLINICAL CASE NUMBER: D1

BREED: Highland AGE 10 yrs SEX:Female DATE OF REFERRAL: 25.2.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oesophagus, oesophageal groove (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Snabhead, Avonbridge, Stirlingshire

HOME BRED OR BOUGHT IN Bought in at 7 years of age

BRACKEN INFESTATION Nil

ACCESS Never in three years on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Farm on Western Isles. Bracken exposure unknown

EPIDEMIOLOGY CASE NUMBER: E3 CLINICAL CASE NUMBER: B3

BREED: Galloway AGE 14 yrs SEX: Female DATE OF REFERRAL: 31.8.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Rumen (1)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Inveryne, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (21% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E4 CLINICAL CASE NUMBER: B5

BREED: Aberdeen Angus X AGE 8 yrs SEX: Female DATE OF REFERRAL: 27.2.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx, oesophagus

UAP Tongue, pharynx, rumen (6)

IAC -

MUBN -

BUBN -

OTHER Melanoma of skin on foetus in utero

FARM OF ORIGIN Failte, Lochgoilhead, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 years of age

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Ardnahein, Lochgoilhead, Argyll. Moderate infestation (15% of pastures). Access at all times.

EPIDEMIOLOGY CASE NUMBER: E5 CLINICAL CASE NUMBER: C1

BREED: Highland X AGE 11 yrs SEX: Female DATE OF REFERRAL: 1.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Tongue, palate, Oesophagus, cardia, oesophageal groove, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E6 CLINICAL CASE NUMBER: D3

BREED: Galloway X AGE 10 yrs SEX: Female DATE OF REFERRAL: 22.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oropharynx, oesophagus, rumen (11)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Borrodale, Arisaig, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (8% of pastures), primarily on hill land where cattle graze

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E7 CLINICAL CASE NUMBER: B6

BREED: Shorthorn AGE 15 yrs SEX: Female DATE OF REFERRAL: 18.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia, oesophageal groove, rumen

UAP Tongue, palate, oesophagus, cardia, oesophageal groove, rumen (> 45)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum, small intestine, large intestine, rectum

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed V.S. and self)

PREVIOUS ORIGINS Corra, Tignabruaich, Argyll. Moderate infestation. Access at all times

EPIDEMIOLOGY CASE NUMBER: E8 CLINICAL CASE NUMBER: C2

BREED: Shorthorn X AGE 13 yrs SEX: Female DATE OF REFERRAL: 18.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia

UAP Palate (1)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Ballinaby, Gruinart, Islay, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E9 CLINICAL CASE NUMBER: ^8

BREED: Highland X AGE >10 yrs SEX: Female DATE OF REFERRAL: 21.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP -

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN High Ronachan, Tarbert, Argyll

HOME BRED OR BOUGHT IN Bought in at > 5 years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Frequent. Occasionally grazed on bracken free pastures

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (Farmer)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E10 CLINICAL CASE NUMBER: -

BREED: Highland AGE 10 yrs SEX: Female DATE OF REFERRAL: 2.5.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, oesophagus (> 15)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Inveryne, Kilfinan, Argylle

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (21% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E11 CLINICAL CASE NUMBER: B7

BREED: Aberdeen Angus AGE 9 yrs SEX: Female DATE OF REFERRAL: 30.5.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Oropharynx, oesophagus, rumen (>20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Carse, Tarbert, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E12 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 12 yrs SEX: Female DATE OF REFERRAL: 29.5.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, oesophageal groove, rumen

UAP Oesophageal groove (3)

IAC -

MUBN -

BUBN -

OTHER Haemangioma of urethra

FARM OF ORIGIN Glenborrodale, Ardnamurchan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E13 CLINICAL CASE NUMBER: C4

BREED: Highland X AGE 15 yrs SEX: Female DATE OF REFERRAL: 9.8.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oesophagus (6)

IAC Caecum and colon

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine. Fibroma of oesophagus.

FARM OF ORIGIN Corranmore, Ardfern, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Frequent. Occasionally grazed on bracken free pastures

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E14 CLINICAL CASE NUMBER: C5

BREED: Highland X AGE 13 yrs SEX: Female DATE OF REFERRAL: 11.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia

UAP Palate, oesophagus (7)

IAC -

MUBN -

BUBN -

OTHER Lipoma of colon

FARM OF ORIGIN Glenborrodale, Ardnamurchan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E15 CLINICAL CASE NUMBER: B8

BREED: ABERDEEN ANGUS AGE 15 yrs SEX: Female DATE OF REFERRAL: 25.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Palate, rumen (> 15)

IAC Jejunum

MUBN -

BUBN -

OTHER Phaeochromocytoma of adrenal gland

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E16 CLINICAL CASE NUMBER: B9

BREED: Aberdeen Angus AGE 18 yrs SEX: Female DATE OF REFERRAL: 23.11.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Palate, rumen (11)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of colon, adenoma of gall bladder

FARM OF ORIGIN Munigierie, Invergarry, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 15 years of age

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Arisaig, Inverness-shire. Infestation moderate (8% of pastures). Access at all times from calfuntil sold.

EPIDEMIOLOGY CASE NUMBER: E17 CLINICAL CASE NUMBER: A10

BREED: Shorthorn AGE 8 yrs SEX: Female DATE OF REFERRAL: 28.11.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, pharynx, oesophagus, rumen

UAP Oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine and colon

FARM OF ORIGIN Fidden, Fionnphort, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (18% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E18 CLINICAL CASE NUMBER: C7

BREED: Aberdeen Angus AGE 10 yrs SEX: Female DATE OF REFERRAL: 22.1.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Palate, pharynx, oesophagus, oesophageal groove

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Kilmichaelbeg, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E19 CLINICAL CASE NUMBER: C8

BREED: Shorthorn X AGE 10 yrs SEX: Female DATE OF REFERRAL: 24.5.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, oesophageal groove

UAP Palate, oesophagus (3)

IAC -

MUBN -

BUBN Fibromas

OTHER Phaeochromocytoma of adrenal gland

FARM OF ORIGIN New Ulva, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E20 CLINICAL CASE NUMBER: B10

BREED: Highland AGE 16 yrs SEX: Female DATE OF REFERRAL: 31.5.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Pharynx (2)

IAC -

MUBN -

BUBN -

OTHER Lipoma of colon

FARM OF ORIGIN Kilbridemore, Glendaruel, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Only after mid-July each year

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Isle of Bute. Bracken infestation and access unknown

EPIDEMIOLOGY CASE NUMBER: E21 CLINICAL CASE NUMBER: A12

BREED: Highland AGE 12 yrs SEX: Female DATE OF REFERRAL: 6.6.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus, rumen

UAP Tongue, palate, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E22 CLINICAL CASE NUMBER: D6

BREED: Shorthorn X AGE 12 yrs SEX: Female DATE OF REFERRAL: 6.6.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Palate, oesophagus, rumen (>20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine.
Adenoma of gall bladder

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E23 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 11 yrs SEX: Female DATE OF REFERRAL: 23.6.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Palate, oesophagus (6)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Carse, Tarbert, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E24 CLINICAL CASE NUMBER: C9

BREED: Highland X AGE 10 yrs SEX: Female DATE OF REFERRAL: 9.7.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oesophagus (4)

IAC -

MUBN -

BUBN -

OTHER Adenoma of renal cortex, adenomatous hyperplasia of small intestine.

FARM OF ORIGIN Ballintyre, Inveraray, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (6% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Benderloch, Oban, Argyll. Bracken infestation and access unknown.

EPIDEMIOLOGY CASE NUMBER: E25 CLINICAL CASE NUMBER: D7

BREED: Shorthorn X AGE 17 yrs SEX: Female DATE OF REFERRAL: 24.7.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia, rumen

UAP Palate, oesophagus (9)

IAC -

MUBN -

BUBN -

OTHER Fibroleiomyoma of uterus, phaeochromocytoma of adrenal gland adenomatous hyperplasia of small intestine

FARM OF ORIGIN Kilmichaelbeg, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E26 CLINICAL CASE NUMBER: C10

BREED: Galloway X AGE 8 yrs SEX: Female DATE OF REFERRAL: 16.8.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia

UAP Palate, rumen (6)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Quinish, Dervaig, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E27 CLINICAL CASE NUMBER: F1

BREED: Highland AGE 16 yrs SEX: Female DATE OF REFERRAL: 2.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Tongue, pharynx, oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (28% of pasture)

ACCESS Frequent throughout life. Occasionally grazed on bracken -free pasture.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E28 CLINICAL CASE NUMBER: C11

BREED: Highland X AGE 9 yrs SEX: Female DATE OF REFERRAL: 3.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Oesophagus, rumen (4)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of bile duct papilla

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E29 CLINICAL CASE NUMBER: B11

BREED: Aberdeen Angus AGE 8 yrs SEX: Female DATE OF REFERRAL: 3.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Tongue, palate, pharynx, oesophagus, rumen (> 30)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 months of age

BRACKEN INFESTATION Severe (60% of pasture)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E30 CLINICAL CASE NUMBER: D8

BREED: Highland X AGE 9 yrs SEX: Female DATE OF REFERRAL: 25.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove

UAP Palate, oesophagus, rumen (> 30)

IAC -

MUBN -

BUBN -

OTHER Fibroma of oesophagus, adenomatous hyperplasia of bile duct papilla.

FARM OF ORIGIN Inveryne, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (21% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E31 CLINICAL CASE NUMBER: -

BREED: Galloway X AGE 14 yrs SEX: Female DATE OF REFERRAL: 1 7.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia, rumen

UAP Palate, oesophagus, cardia (> 30)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E32 CLINICAL CASE NUMBER: C12

BREED: Aberdeen Angus X AGE 14 yrs SEX: Female DATE OF REFERRAL: 25.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, rumen

UAP Tongue, palate, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Lergychonybeg, Barbreck, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (22% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Calves (confirmed v.s.)

PREVIOUS ORIGINS Lergychonymore, Barbreck, Argyll. Moderate infestation. Access at all times.

EPIDEMIOLOGY CASE NUMBER: E33 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 9 yrs SEX: Female DATE OF REFERRAL: 25.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Oropharynx, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E34 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 9 yrs SEX: Female DATE OF REFERRAL: 25.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Palate, oesophagus (> 15)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E35 CLINICAL CASE NUMBER: -

BREED: Highland AGE 12 yrs SEX: Female DATE OF REFERRAL: 12.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, oesophagus, oesophageal groove (> 20)

IAC Ileum

MUBN -

BUBN -

OTHER Squamous cell carcinoma of vagina, papillomas of perineum

FARM OF ORIGIN West Torrie, Callander, Perthshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E36 CLINICAL CASE NUMBER: D9

BREED: Aberdeen Angus X AGE 11 yrs SEX: Female DATE OF REFERRAL: 19.4.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophageal groove, rumen

UAP Tongue, palate, pharynx, oesophagus, cardia (> 25)

IAC -

MUBN -

BUBN -

OTHER Papillomas of teats, adenomatous hyperplasia of duodenum

FARM OF ORIGIN Lerags, Oban, Argyll

HOME BRED OR BOUGHT IN Bought in at 9 months of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E37 CLINICAL CASE NUMBER: A15

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 28.5.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, pharynx, oesophagus

UAP Palate, oesophagus, rumen (> 15)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Glenview, Duisky, Argyll

HOME BRED OR BOUGHT IN Bought in at > 10 years of age

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown, purchased at market

EPIDEMIOLOGY CASE NUMBER: E38 CLINICAL CASE NUMBER: C13

BREED: Highland X AGE 9 yrs SEX: Female DATE OF REFERRAL: 9.6.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, pharynx (> 5)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Upper Largie, Kilmartin, Argyll

HOME BRED OR BOUGHT IN Home Bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E39 CLINICAL CASE NUMBER: A16

BREED: Aberdeen Angus AGE 10 yrs SEX: Female DATE OF REFERRAL: 15.6.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, palate, pharynx, oesophagus, rumen

UAP Palate, rumen (6)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Swordle, Acharacle, Argyll

HOME BRED OR BOUGHT IN Bought in at 5 years of age

BRACKEN INFESTATION Severe (22% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Glenborrodale, Ardnamurchan, Argyll. Infestation light (3% of pastures). Access continuous from calf until sold (See E12 and E14)

EPIDEMIOLOGY CASE NUMBER: E40 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 8.12.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Oropharynx, oesophagus, cardia (> 10)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E41 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 11 yrs SEX: Female DATE OF REFERRAL: 23.1.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Oesophagus (1)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Ashfield, Achnamara, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Virtu ally continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E42 CLINICAL CASE NUMBER: -

BREED: Highland AGE > 10 yrs SEX: Female DATE OF REFERRAL: 27.2.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Pharynx, oesophagus (> 10)

IAC Jejunum

MUBN -

BUBN -

OTHER Phaeochromocytoma of adrenal gland, adenomatous hyperplasia of colon.

FARM OF ORIGIN Kilmalieu, Ardgour, Argyll

HOME BRED OR BOUGHT IN Bought in at > 10 years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Knockvologin, Fionnphort, Mull, Argyll. Moderate infestation. Access continuous from calf until sold

EPIDEMIOLOGY CASE NUMBER: E43 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 29.2.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Carse, Tarbert, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E44 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 15 yrs SEX: Female DATE OF REFERRAL: 15.6.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx, oesophagus

UAP Oesophagus, rumen (11)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Moy, Banavie, Fort William, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 13 years of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Drimnatorrin, Strontian, Argyll. Moderate infestation (17% of pastures). Access continuous from calf until sold. (see E113)

EPIDEMIOLOGY CASE NUMBER: E45 CLINICAL CASE NUMBER: F3

BREED: Aberdeen Angus AGE 10 yrs SEX: Female DATE OF REFERRAL: 28.7.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Lower gum and lip

UAP Tongue, pharynx (> 10)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Swordle, Acharacle, Argyll

HOME BRED OR BOUGHT IN Bought in at 5 years of age

BRACKEN INFESTATION Severe (22% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Glenborrodale, Ardnamurchan, Argyll. Infestation light (3% of pastures). Access continuous from calf until sold (See E12 and E14)

EPIDEMIOLOGY CASE NUMBER: E46 CLINICAL CASE NUMBER: D10

BREED: Shorthorn X AGE 8 yrs SEX: Female DATE OF REFERRAL: 27.8.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus

UAP Pharynx, rumen (5)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Upper Largie, Kilmartin, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pasture)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s.

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E47 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 11.9.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, oesophagus, oesophageal groove

UAP Palate, pharynx, oesophagus, rumen (> 25)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Blairour, Spean Bridge, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E48 CLINICAL CASE NUMBER: D11

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 11.9.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oesophagus, rumen (> 25)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Leukary, Kilmichael, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pasture)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E49 CLINICAL CASE NUMBER: D12

BREED: Hereford X AGE 14 yrs SEX: Female DATE OF REFERRAL: 5.11.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia, rumen

UAP Palate, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Carloonan, Inveraray, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (10% of pasture)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Local farm. Bracken infested. Continuous access

EPIDEMIOLOGY CASE NUMBER: E50 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 9 yrs SEX: Female DATE OF REFERRAL: 30.8.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, rumen

UAP Tongue, palate, oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent throughout life. Occasionally grazed on bracken -free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults, (confirmed v.s. and self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E51 CLINICAL CASE NUMBER: -

BREED: Galloway X AGE 8 yrs SEX: Female DATE OF REFERRAL: 16.9.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue

UAP Palate, oesophagus, rumen (> 15)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Killiechronan, Salen, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (14% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E52 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 16.12.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophageal groove, rumen

UAP Palate,oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Kilmichaelbeg, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E53 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 13 yrs SEX: Female DATE OF REFERRAL: 14.1.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus, oesophageal groove

UAP Tongue, palate, pharynx, oesophagus, rumen (> 35)

IAC -

MUBN -

BUBN -

OTHER Adenoma of bile duct

FARM OF ORIGIN Hyndhope, Ettrick Bridge, Selkirkshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E54 CLINICAL CASE NUMBER: -

BREED: Highland X AGE 18 yrs SEX: Female DATE OF REFERRAL: 25.1.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Palate, oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Escart, Tarbert, Argyll

HOME BRED OR BOUGHT IN Bought in at one month of age

BRACKEN INFESTATION Severe (24% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Calf (Farmer)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E55 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 15.2.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, oesophageal groove

UAP Pharynx, oesophagus (> 10)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Dalry, Kirkton, Taynuilt, Argyll

HOME BRED OR BOUGHT IN Bought in at one year of age

BRACKEN INFESTATION Severe (> 20% of pastures). Common hill grazir

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Oban market

EPIDEMIOLOGY CASE NUMBER: E56 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 16.6.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, oesophageal groove, rumen

UAP Oesophagus, rumen (4)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent throughout life. Occasionally grazed on bracken-free pastures

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E57 CLINICAL CASE NUMBER: -

BREED: Highland AGE 15 yrs SEX: Female DATE OF REFERRAL: 19.6.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, oesophagus

UAP Pharynx, oesophagus, rumen (> 30)

IAC -

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of colon

FARM OF ORIGIN Gartincaber, Balmaha, Stirlingshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Virtually continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E58 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 9 yrs SEX: Female DATE OF REFERRAL: 31.8.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, cardia, rumen

UAP Tongue, palate, pharynx, oesophagus, rumen (> 75)

IAC Jejunum

MUBN -

BUBN -

OTHER Adenomatous hyperplasia of small intestine, colon, rectum

FARM OF ORIGIN The Store, Onich, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures). Common grazing

ACCESS Continuous throughout life except when housed October - May

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E59 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 14.2.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP -

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Kinkell, Lennoxtown, Dumbartonshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (2% of pastures). All on rented pasture off main farm.

ACCESS Only access possible as a heifer. No access after 3 years of age when entered dairy herd.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E60 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 10 yrs SEX: Female DATE OF REFERRAL: 8.3.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophageal groove

UAP -

IAC -

MUBN -

BUBN -

OTHER Fibropapilloma of teat

FARM OF ORIGIN Balnalachlan, Callander, Perthshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Continuous throughout life except when housed in winter.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, adults (confirmed v.s. ar self).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E61 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 12 yrs SEX: Female DATE OF REFERRAL: 23.3.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx, rumen

UAP Pharynx, oesophagus (5)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Balnalachlan, Callander, Perthshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Continuous throughout life except when housed in winter.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, adults (confirmed v.s. and self).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E62 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 11 yrs SEX: Female DATE OF REFERRAL: 27.5.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, oesophagus (5)

IAC Jejunum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Smirsary, Glenuig, Lochailort, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E63 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 11 yrs SEX: Female DATE OF REFERRAL: 23.7.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx

UAP -

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Kilmichaelbeg, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E64 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 8 yrs SEX: Female DATE OF REFERRAL: 30.8.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate (1)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Upper Largie, Kilmartin, Argyll

HOME BRED OR BOUGHT IN Bought in at 7 years of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmedv.s.)

PREVIOUS ORIGINS Keel Farm, Tayvallich, Argyll. Bracken infested. Continuous access from calf until sold.

EPIDEMIOLOGY CASE NUMBER: E65 CLINICAL CASE NUMBER: A14

BREED: Aberdeen Angus AGE 12 yrs SEX: Female DATE OF REFERRAL: 29.1.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Pharynx

UAP Palate, pharynx, oesophagus, rumen (11)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Christcliffe, Boot, Eskdale, Cumbria

HOME BRED OR BOUGHT IN Bought in at 9 years of age

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Local farm dissolution sale. Severe bracken infestation. Continuous access from calf until sold.

EPIDEMIOLOGY CASE NUMBER: E66 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 8 yrs SEX: Female DATE OF REFERRAL: 10.12.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, pharynx (> 10)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN West Browncastle, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 6 years of age

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E67 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 6.1.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Tongue, pharynx, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Ugie Brae, Strichen, Aberdeenshire

HOME BRED OR BOUGHT IN Bought in at > 10 years of age in spring 197

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS Unknown. Ear tag indicates originated in Inverness-shire.

EPIDEMIOLOGY CASE NUMBER: E68 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 13 yrs SEX: Female DATE OF REFERRAL: 1.2.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Pharynx, oesophagus (6)

IAC -

MUBN -

BUBN Haemangioma

OTHER -

FARM OF ORIGIN Boreland of Southwick, Dalbeattie, Kirkcudbright

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous when on farm of origin except when yarded October to March.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS Ireland

EPIDEMIOLOGY CASE NUMBER: E69 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 17 yrs SEX: Female DATE OF REFERRAL: 26.1.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus

UAP Tongue, rumen (3)

IAC -

MUBN Transitional cell carcinoma, adenocarcinoma

BUBN Haemangiomas

OTHER Adenomas of colon, haemangiomas of endometrium

FARM OF ORIGIN Lower Dunain, Inverness, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Frequent

INCIDENTS OF ACUTE BRACKEN POISONING Calf (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E70 CLINICAL CASE NUMBER: CB4

BREED: Shorthorn X AGE 13 yrs SEX: Female DATE OF REFERRAL: 11.2.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, oesophagus, rumen

UAP Tongue, palate, pharynx, rumen (> 20)

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Carse, Tarbert, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E71 CLINICAL CASE NUMBER: D2

BREED: Galloway AGE 10 yrs SEX: Female DATE OF REFERRAL: 21.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Palate, oesophagus, rumen (> 10)

IAC -

MUBN -

BUBN Haemangiomas

OTHER Adenomas of duodenum

FARM OF ORIGIN Glenside, Kirkmichael, Ayrshire

HOME BRED OR BOUGHT IN Bought in at > 10 years of age

BRACKEN INFESTATION Nil

ACCESS Never when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Newton Stewart area, Kirkcudbright.

EPIDEMIOLOGY CASE NUMBER: E72 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 9 yrs SEX: Female DATE OF REFERRAL: 2.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, rumen

UAP Palate, oesophagus (> 20)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Banlicken, Pirnmill, Arran, Bute

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Port William area, Wigtownshire

EPIDEMIOLOGY CASE NUMBER: E73 CLINICAL CASE NUMBER: D4

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 11.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophageal groove, rumen

UAP Tongue, palate, pharynx (> 20)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Ardnaw, Achnamara, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E74 CLINICAL CASE NUMBER: -

BREED: Highland X AGE 12 yrs SEX: Female DATE OF REFERRAL: 11.6.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, oesphagus

UAP Oesophagus (> 20)

IAC -

MUBN -

BUBN Haemangioma

OTHER Adenomas of small intestine

FARM OF ORIGIN Balliemore Estates, Otter Ferry,Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Occasional

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (Farmer)

PREVIOUS ORIGINS Unknown. Purchased at Oban market.

EPIDEMIOLOGY CASE NUMBER: E75 CLINICAL CASE NUMBER: C3

BREED: Aberdeen Angus AGE 12 yrs SEX: Female DATE OF REFERRAL: 22.6.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Cardia, rumen

UAP Palate, pharynx (13)

IAC -

MUBN Haemangiosarcoma

BUBN -

OTHER Adenomas of small intestine

FARM OF ORIGIN Balure, Lismore, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E76 CLINICAL CASE NUMBER: -

BREED: Highland X AGE 14 yrs SEX: Female DATE OF REFERRAL: 29.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophageal groove at cardia

UAP Tongue, palate, pharynx, oesophagus, cardia (> 20)

IAC Jejunum

MUBN -

BUBN Haemangiomas

OTHER Adenoma of duodenal papilla

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent when on farm of origin. Occasionally grazed on bracken-free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.and self)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E77 CLINICAL CASE NUMBER: A9

BREED: Highland AGE 14 yrs SEX: Female DATE OF REFERRAL: 28.11.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Palate, oesophagus, rumen

UAP Oesophagus (21)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER Adenoma of renal cortex, melanoma of pelvic connective tissu

FARM OF ORIGIN Killunaig, Pennyghael, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (23% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E78 CLINICAL CASE NUMBER: U7

BREED: Aberdeen Angus AGE 8 yrs SEX: Female DATE OF REFERRAL: 14.12.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, oesophageal groove, rumen

UAP Oesophagus, rumen (11)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Adenomas and adenomatous hyperplasia of duodemum, colon and rectum.

FARM OF ORIGIN Borrodale, Arisaig, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (7% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E79 CLINICAL CASE NUMBER: D5

BREED: Aberdeen Angus AGE 13 yrs SEX: Female DATE OF REFERRAL: 17.5.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, oesophagus, oesphageal groove, rumen

UAP Palate, pharynx, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN Fibroma

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s. and self).

PREVIOUS ORIGINS Unknown. Purchased from cattle dealer.

EPIDEMIOLOGY CASE NUMBER: E80 CLINICAL CASE NUMBER: -

BREED: Highland X AGE 10 yrs SEX: Female DATE OF REFERRAL: 25.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Oesophagus (10)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER Lipomas of rumen, adenomatous hyperplasia of colon

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E81 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 8 yrs SEX: Female DATE OF REFERRAL: 16.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, cardia, rumen

UAP Tongue, palate, oesophagus, rumen (> 25)

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Caddleton, Balvicar, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (43% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS and Luing, Argyll. Bracken infestation in island severe access highly probable from calf until sold.

EPIDEMIOLOGY CASE NUMBER: E82 CLINICAL CASE NUMBER: U18

BREED: Ayrshire AGE 9 yrs SEX: Female DATE OF REFERRAL: 11.5.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Oesophagus, rumen

UAP Palate, pharynx, oesophagus, rumen (> 15)

IAC Duodenum

MUBN -

BUBN Haemangiomas, fibromas

OTHER Adenoma of thyroid, adenoma of cytic duct of gall bladder, adenomatous hyperplasia of duodenum and colon

FARM OF ORIGIN Kilbride, Kilmore, Oban, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (8% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E83 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 11 yrs SEX: Female DATE OF REFERRAL: 20.7.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Tongue, pharynx, cardia

UAP Palate, oesophagus, cardia (> 20)

IAC -

MUBN -

BUBN Haemangioma

OTHER Fibroma of rumen, adenomatous hyperplasia of small intestine

FARM OF ORIGIN Carry, Ardlamont, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E84 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 7 yrs SEX: Female DATE OF REFERRAL: 30.12.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC Rumen

UAP Palate, pharynx (> 15)

IAC -

MUBN -

BUBN Haemangioma

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Achara, Duror, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (7% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

APPENDIX 4.2

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA OF ANIMALS WITH UPPER ALIMENTARY PAPILLOMAS. (Animals which were also affected by malignant neoplasia are recorded in Appendices 4.1, 4.3, 4.4, 4.5, 4.6).

EPIDEMIOLOGY CASE NUMBER: E85

BREED: Ayrshire AGE: 3½ yrs SEX: Female DATE OF REFERENCE: 14.6.72

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Commonside, Annbank, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Nil

ACCESS Never when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased from cattle dealer in Galston, Ayrshire.

EPIDEMIOLOGY CASE NUMBER: E86

BREED: Ayrshire AGE: 4 yrs SEX: Female DATE OF REFERENCE: 4.1.73

UAP (SITE AND NUMBER) Oesophagus (3)

FARM OF ORIGIN Haldykes, Lockerbie, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E87

BREED: Shorthorn X AGE: 10 yrs SEX: Femal DATE OF REFERENCE: 10.3.73

UAP (SITE AND NUMBER) Tongue, palate, rumen (5)

FARM OF ORIGIN Ederline, Ford, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Frequent. Also grazed bracken free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E88

BREED: Shorthorn AGE: 10 yrs SEX: Femal DATE OF REFERENCE: 23.3.73

UAP (SITE AND NUMBER) Palate (1)

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent. Occasionally grazed on bracken-free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E89

BREED: Ayrshire AGE: 9 yrs SEX: Female DATE OF REFERENCE: 27.4.73

UAP (SITE AND NUMBER) Oropharynx, oesophagus, rumen (5)

FARM OF ORIGIN Ballinaby, Gruinart, Islay, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous until first calving. Thereafter access very infrequent.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E90

BREED: Shorthorn AGE: 3 yrs SEX: Female DATE OF REFERENCE: 6.6.73

UAP (SITE AND NUMBER) Rumen (1)

FARM OF ORIGIN Milton, Kirkcudbright, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E91

BREED: Aberdeen Angus AGE: 1½ yrs SEX: Female DATE OF REFERENCE: 17.7.73

UAP (SITE AND NUMBER) Palate, oesophagus (4)

FARM OF ORIGIN Oak Croft, Onich, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS April to October each year. Housed in winter.

INCIDENTS OF ACUTE BRACKEN POISONING This animal (confirmed self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E92

BREED: Ayrshire AGE: 10 yrs SEX: Female DATE OF REFERENCE: 17.8.73

UAP (SITE AND NUMBER) Oesophagus (7)

FARM OF ORIGIN Glenreasdell, Whitehouse, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Frequent between April and October. Housed in winter.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E93

BREED: Hereford AGE: 6 yrs SEX: Female DATE OF REFERENCE: 23.8.73

UAP (SITE AND NUMBER) Oropharynx (5)

FARM OF ORIGIN Brenchoille, Furnace, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E94

BREED: Shorthorn AGE: 12 yrs SEX: Female DATE OF REFERENCE: 4.11.73

UAP (SITE AND NUMBER) Pharynx, oesophagus, rumen (10)

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s. and self).

PREVIOUS ORIGINS Unknown. Aberdeenshire.

EPIDEMIOLOGY CASE NUMBER: E95

BREED: Hereford AGE: 6 yrs SEX: Male DATE OF REFERENCE: 15.11.73

UAP (SITE AND NUMBER) Pharynx (1)

FARM OF ORIGIN Monachylemore, Balquhidder, Perthshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Perth market.

EPIDEMIOLOGY CASE NUMBER: E96

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 22.11.73

UAP (SITE AND NUMBER) Rumen (6)

FARM OF ORIGIN Walton, Castlecary, Stirlingshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasional, but only as heifer.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E97

BREED: Welsh Black AGE: 10 yrs SEX: Female DATE OF REFERENCE: 23.11.73

UAP (SITE AND NUMBER) Oropharynx, oesophagus, rumen (8)

FARM OF ORIGIN Garray Gualach, Invergarry, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Ceunant, North Wales.

EPIDEMIOLOGY CASE NUMBER: E98

BREED: Galloway X AGE: 5 yrs SEX: Female DATE OF REFERENCE: 24.11.73

UAP (SITE AND NUMBER) Palate, Oesophagus (>10)

FARM OF ORIGIN Mark, Creetown, Wigtownshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Frequent, particularly during summer months

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E99

BREED: Shorthorn AGE: 18 yrs SEX: Female DATE OF REFERENCE: 4.12.73

UAP (SITE AND NUMBER) Oropharynx, rumen (4)

FARM OF ORIGIN Stronmagachan, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E100

BREED: Aberdeen Angus X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 9.2.74

UAP (SITE AND NUMBER) Oropharynx (6)

FARM OF ORIGIN Melford, Kilmelford, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (31% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING This animal (confirmed self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E101

BREED: Shorthorn X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 19.2.74

UAP (SITE AND NUMBER) Oropharynx (4)

FARM OF ORIGIN Midglen, Crossford, Moniave, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (33% of pastures)

ACCESS Continuous October to March, occasionally during summer months.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS Unknown. Ireland.

EPIDEMIOLOGY CASE NUMBER: E102

BREED: Shorthorn X AGE: 12 yrs SEX: Female DATE OF REFERENCE: 16.8.74

UAP (SITE AND NUMBER) Tongue, oesophagus (3)

FARM OF ORIGIN Moy, Banavie, Fort William, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 6 years of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous when on farm of origin.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Corpach market.

EPIDEMIOLOGY CASE NUMBER: E103

BREED: Aberdeen Angus AGE: 14 yrs SEX: Female DATE OF REFERENCE: 30.8.74

UAP (SITE AND NUMBER) Oropharynx (8)

FARM OF ORIGIN Corranbeg, Ardfern, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 months of age

BRACKEN INFESTATION Severe (34% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Reara, Oban, Argyllshire. Bracken infested.

EPIDEMIOLOGY CASE NUMBER: E104

BREED: Shorthorn X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 2.9.74

UAP (SITE AND NUMBER) Oropharynx, oesophagus (6)

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent throughout life. Occasionally grazed on bracken-free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS Unknown.

EPIDEMIOLOGY CASE NUMBER: E105

BREED: Shorthorn AGE: 8 yrs SEX: Female DATE OF REFERENCE: 13.9.74

UAP (SITE AND NUMBER) Oesophagus (4)

FARM OF ORIGIN Ardnaw, Achnamara, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E106

BREED: Shorthorn X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 17.10.74

UAP (SITE AND NUMBER) Palate, oesophagus (13)

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (23% of pastures)

ACCESS Frequent throughout life. Occasionally grazed on bracken-free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E107

BREED: Hereford X AGE: 9 yrs SEX: Female DATE OF REFERENCE: 17.10.74

UAP (SITE AND NUMBER) Tongue, palate, oesophagus (> 20)

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E108

BREED: Shorthorn X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 25.10.74

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E109

BREED: Hereford X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 19.11.74

UAP (SITE AND NUMBER) Palate (> 10)

FARM OF ORIGIN Cross, Morar, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Ireland.

EPIDEMIOLOGY CASE NUMBER: E110

BREED: Shorthorn X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 2.12.74

UAP (SITE AND NUMBER) Oropharynx (6)

FARM OF ORIGIN Barbreck, Kilchrennan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (8% of pastures)

ACCESS Occasional throughout life when grazed on hill pastures.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Doon, Oban, Argyll. Bracken infested.

EPIDEMIOLOGY CASE NUMBER: E111

BREED: Shorthorn X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 6.1.75

UAP (SITE AND NUMBER) Palate (3)

FARM OF ORIGIN Auchenlochan, Tignabruiach, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (6% of pastures)

ACCESS April to October every year

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E112

BREED: Ayrshire AGE: 10 yrs SEX: Female DATE OF REFERENCE: 23.1.75

UAP (SITE AND NUMBER) Palate (1)

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 weeks of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s. and self).

PREVIOUS ORIGINS Unknown. Purchased at Paisley market.

EPIDEMIOLOGY CASE NUMBER: E113

BREED: Galloway X AGE: 12 yrs SEX: Female DATE OF REFERENCE: 6.2.75

UAP (SITE AND NUMBER) Palate, oesophagus (5)

FARM OF ORIGIN Drimnatorran, Strontian, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E114

BREED: Hereford AGE: 17 yrs SEX: Female DATE OF REFERENCE: 25.2.75

UAP (SITE AND NUMBER) Tongue, palate, oesophagus (8)

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 3½ years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Continuous on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Yes. Heifers, adults

PREVIOUS ORIGINS Bealach-an-drain, Glendaruel, Argyll. Moderate bracken infestation (14% of pastures) (See E14).

EPIDEMIOLOGY CASE NUMBER: E115

BREED: Shorthorn X AGE: 4yrs SEX: Female DATE OF REFERENCE: 28.3.75

UAP (SITE AND NUMBER) Palate (1)

FARM OF ORIGIN Achmacarry, Gairlochy, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Light (1% of pastures)

ACCESS Frequently when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Dalilee, Ardnamurchan, Argyll. Bracken infested.

EPIDEMIOLOGY CASE NUMBER: E116

BREED: Hereford X AGE: 4 yrs SEX: Female DATE OF REFERENCE: 8.8.75

UAP (SITE AND NUMBER) Palate, oesphagus (6)

FARM OF ORIGIN Glencalvie, Ardgay, Ross and Cromarty

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (38% of pastures)

ACCESS Continuous throughout life. Particularly when on hill grazing in summer.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults including this animal (confirmed self).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E117

BREED: Shorthorn X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 8.8.75

UAP (SITE AND NUMBER) Oesophagus (4)

FARM OF ORIGIN Glencalvie, Ardgay, Ross and Cromarty

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Severe (38% of pastures)

ACCESS Continuous throughout life. Particularly when on hill grazing in summer.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults including this animal (confirmed self)

PREVIOUS ORIGINS Unknown. Local farm.

EPIDEMIOLOGY CASE NUMBER: E118

BREED: Galloway X AGE: 9 yrs SEX: Female DATE OF REFERENCE: 13.10.75

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Auchencar, Machrie, Arran, Bute

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (50% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adult (Farmer)

PREVIOUS ORIGINS Unknown. Purchased at Brodick Market, Arran.

EPIDEMIOLOGY CASE NUMBER: E119

BREED: Galloway AGE: 9 yrs SEX: Female DATE OF REFERENCE: 7.11.75

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Woodneuk, Barrhead, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (8% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E120

BREED: Galloway AGE: 13 yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Palate, Oesophagus (2)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed vs.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E121

BREED: Shorthorn X AGE: 9yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Tongue, oesophagus (2)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E122

BREED: Shorthorn X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E123

BREED: Galloway AGE: 13 yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Palate, oeophagus (12)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E124

BREED: Galloway AGE: 13 yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E125

BREED: Shorthorn X AGE: 9 yrs SEX: Female DATE OF REFERENCE: 8.12.75

UAP (SITE AND NUMBER) Palate, oesophagus, rumen (> 20)

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E126

BREED: Aberdeen Angus AGE: 4 yrs SEX: Female DATE OF REFERENCE: 16.1.76

UAP (SITE AND NUMBER) Palate, oesophagus (2)

FARM OF ORIGIN Dunain Mains, Inverness, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Frequently throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E127

BREED: Shorthorn X AGE: 1½ yrs SEX: Female DATE OF REFERENCE: 10.2.76

UAP (SITE AND NUMBER) Oropharynx, oesophagus (> 10)

FARM OF ORIGIN Bailliemeanach, Cladich, Argyll

HOME BRED OR BOUGHT IN -Home bred

BRACKEN INFESTATION Severe (42% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E128

BREED: Hereford X AGE: 12 yrs SEX: Female DATE OF REFERENCE: 16.9.76

UAP (SITE AND NUMBER) Palate (4)

FARM OF ORIGIN Galloichoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E129

BREED: Friesian AGE: 8 yrs SEX: Female DATE OF REFERENCE: 21.10.76

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Wester Campfield, Glassel, Banchory, Aberdeenshire

HOME BRED OR BOUGHT IN Bought in at 3 days of age

BRACKEN INFESTATION Moderate (9% of pastures)

ACCESS April to October each year

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown.

EPIDEMIOLOGY CASE NUMBER: E130

BREED: Shorthorn X AGE: 13 yrs SEX: Female DATE OF REFERENCE: 11.1.77

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Killinochnoch, Lochgilphead, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Moderate (8% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E131

BREED: Hereford AGE: 6 yrs SEX: Female DATE OF REFERENCE: 13.1.77

UAP (SITE AND NUMBER) Oesophagus (3)

FARM OF ORIGIN West Glenbuck, Douglas, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 1 week of age

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Lanark market.

EPIDEMIOLOGY CASE NUMBER: E132

BREED: Shorthorn AGE: 10 yrs SEX: Female DATE OF REFERENCE: 21.1.77

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Balliemeanach, Gribun, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E133

BREED: Galloway X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 27.1.77

UAP (SITE AND NUMBER) Tongue, palate, oesophagus (7)

FARM OF ORIGIN Gartnagrenach, Whitehouse, Argyll

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Light (4% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Ireland.

EPIDEMIOLOGY CASE NUMBER: E134

BREED: Shorthorn X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 28.1.77

UAP (SITE AND NUMBER) Tongue (1)

FARM OF ORIGIN Roberton, Borgue, Kirkcudbright.

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (confirmed v.s)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E135

BREED: Galloway AGE: 17 yrs SEX: Female DATE OF REFERENCE: 28.2.77

UAP (SITE AND NUMBER) Tongue (1)

FARM OF ORIGIN Skipness Est., Tarbert, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E136

BREED: Highland AGE: 11 yrs SEX: Female DATE OF REFERENCE: 2.3.77

UAP (SITE AND NUMBER) Tongue, palate, oesophagus (25)

FARM OF ORIGIN Tullich, Kilmelford, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 months of age

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E137

BREED: Highland AGE: 8 yrs SEX:Female DATE OF REFERENCE: 23.3.77

UAP (SITE AND NUMBER) Palate, pharynx (6)

FARM OF ORIGIN Tullich, Kilmelford, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E138

BREED: Aberdeen Angus X AGE:7 yrs SEX:Female DATE OF REFERENCE: 28.4.77

UAP (SITE AND NUMBER) Tongue, palate, pharynx,oesophagus, rumen (7)

FARM OF ORIGIN Ardmarnock, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (28% of pastures)

ACCESS Frequent. Occasionally grazed on bracken-free pastures.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s. and self)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E139

BREED: Shorthorn X AGE: 15 yrs SEX: Female DATE OF REFERENCE: 9.6.77

UAP (SITE AND NUMBER) Oesophagus (3)

FARM OF ORIGIN Grassfield, Whitehouse, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E140

BREED: Shorthorn X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 21.10.77

UAP (SITE AND NUMBER) Palate (3)

FARM OF ORIGIN Cowford, Carstairs, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Hardington Mains, Lamington, Lanarkshire.

EPIDEMIOLOGY CASE NUMBER: E141

BREED: Hereford X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 21.11.77

UAP (SITE AND NUMBER) Palate (3)

FARM OF ORIGIN Bealach-an-drain, Glendaruel,Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (14% of pastures)

ACCESS Occasional but particularly in autumn when on hill grazing.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E142

BREED: Shorthorn AGE: 8 yrs SEX: Female DATE OF REFERENCE: 17.2.78

UAP (SITE AND NUMBER) Oropharynx, oesophagus, rumen (> 30)

FARM OF ORIGIN Grassfield, Whitehouse, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E143

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 4.3.78

UAP (SITE AND NUMBER) Pharynx (2)

FARM OF ORIGIN Park, Auldhouse, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E144

BREED: Hereford X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 2.4.78

UAP (SITE AND NUMBER) Pharynx, oesophagus (2)

FARM OF ORIGIN Midglen, Crossford, Moniave, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Severe (33% of pastures)

ACCESS Continuous October to March. Occasionally during summer mon

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS Unknown. Ireland.

EPIDEMIOLOGY CASE NUMBER: E145

BREED: Hereford AGE: 16 yrs SEX: Female DATE OF REFERENCE: 1.11.78

UAP (SITE AND NUMBER) Rumen (1)

FARM OF ORIGIN Cleghorn Est., Forth, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Light (1% of pastures)

ACCESS Very rare

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Lanark market.

EPIDEMIOLOGY CASE NUMBER: E146

BREED: Shorthorn AGE: 9 yrs SEX: Female DATE OF REFERENCE: 21.3.79

UAP (SITE AND NUMBER) Oropharynx (1)

FARM OF ORIGIN Acharacle, Bunessan, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E147

BREED: Highland AGE: 18 yrs SEX: Female DATE OF REFERENCE: 8.4.79

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Killundine, Drimnin, Morven, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E148

BREED: Aberdeen Angus AGE: 7 yrs SEX: Female DATE OF REFERENCE: 3.12.74

UAP (SITE AND NUMBER) Oropharynx (1)

FARM OF ORIGIN West Dunnmahill, North Stainmore, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Occasional. Approximately 4 months each summer

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E149

BREED: Galloway X AGE: 5 yrs SEX: Female DATE OF REFERENCE: 20.2.75

UAP (SITE AND NUMBER) Palate (3)

FARM OF ORIGIN Everard, Lowick, Cumbria

HOME BRED OR BOUGHT IN Bought in at 2 weeks of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E150

BREED: Friesian AGE: 4 yrs SEX: Female DATE OF REFERENCE: 20.5.72

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Barrhill, Mauchline, Ayrshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E151

BREED: Shorthorn AGE: 14 yrs SEX: Female DATE OF REFERENCE: 24.1.73

UAP (SITE AND NUMBER) Palate, pharynx, oesophagus (4)

FARM OF ORIGIN Kilbride, Balvicar, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E152

BREED: Shorthorn AGE: 9 yrs SEX: Female DATE OF REFERENCE: 29.3.73

UAP (SITE AND NUMBER) Oropharynx (3)

FARM OF ORIGIN Killean, Lismore, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E153

BREED: Shorthorn X AGE: 10yrs SEX: Female DATE OF REFERENCE: 28.4.73

UAP (SITE AND NUMBER) Oropharynx, oesophagus, cardia (> 20)

FARM OF ORIGIN Lephinmore, Saddel, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E154

BREED: Highland X AGE: 10yrs SEX: Female DATE OF REFERENCE: 9.8.73

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Kinlochlaigh,Appin, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E155

BREED: Ayrshire AGE: Unknown SEX: Female DATE OF REFERENCE: 24.11.73

UAP (SITE AND NUMBER) Oesophagus (1)

FARM OF ORIGIN Hall Barns, Kilmarnock, Ayrshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E156

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 6.1.74

UAP (SITE AND NUMBER) Oesophagus (2)

FARM OF ORIGIN Barr Hill, Lennoxtown, Dunbartonshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E157

BREED: Galloway AGE: 9 yrs SEX: Female DATE OF REFERENCE: 14.4.75

UAP (SITE AND NUMBER) Palate (4)

FARM OF ORIGIN Kinlochlaigh, Appin, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E158

BREED: Highland X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 24.10.75

UAP (SITE AND NUMBER) Oesophagus (3)

FARM OF ORIGIN High Ugadale, Carradale, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E159

BREED: Shorthorn X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 14.376

UAP (SITE AND NUMBER) Palate (1)

FARM OF ORIGIN Balgunloune, North Kessock, Inverness

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E160

BREED: Friesian AGE: 1½yrs SEX: Male DATE OF REFERENCE: 19.5.74

UAP (SITE AND NUMBER) Rumen (1)

FARM OF ORIGIN Lanslea, Lochwinnoch, Renfrewshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

APPENDIX 4.3

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA
OF ANIMALS WITH INTESTINAL ADENOCARCINOMA
(Animals which were also affected by upper alimentary squamous cell carcinoma or malignant urinary bladder neoplasia are recorded in Appendices 4.1 and 4.4).

EPIDEMIOLOGY CASE NUMBER: E161 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 7 yrs SEX: Female DATE OF REFERRAL: 6.6.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC Duodenum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Nether Boreland, Boreland, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 5 years of age

BRACKEN INFESTATION Nil

ACCESS Never when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Blacklaw, Beattock, Lockerbie. Now planted by Forestry Commission. Almost certainly was bracken infested.

EPIDEMIOLOGY CASE NUMBER: E162 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 29.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (1)

IAC Jejunum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Kilchamaig, Whitehouse, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E163 CLINICAL CASE NUMBER: -

BREED: Friesian AGE 6 yrs SEX: Female DATE OF REFERRAL: 1.11.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC Jejunum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Shovelboard, Houston, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Frequent

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E164 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 9.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC Jejunum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Roberton, Borgue, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Frequent as calf and heifer but seldom after joining dairy herd.

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E165 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 10 yrs SEX: Female DATE OF REFERRAL: 30.9.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Oesophagus (8)

IAC Jejunum

MUBN -

BUBN -

OTHER -

FARM OF ORIGIN Leukary, Kilmichael, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: CLINICAL CASE NUMBER:

BREED: AGE SEX: DATE OF REFERRAL:

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC

UAP

IAC

MUBN

BUBN

OTHER

FARM OF ORIGIN

HOME BRED OR BOUGHT IN

BRACKEN INFESTATION

ACCESS

INCIDENTS OF ACUTE BRACKEN POISONING

PREVIOUS ORIGINS

APPENDIX 4.4

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA OF ANIMALS WITH MALIGNANT URINARY BLADDER NEOPLASMS.

(Animals which were also affected by upper alimentary squamous cell carcinoma are recorded in Appendix 4.1).

EPIDEMIOLOGY CASE NUMBER: E166 CLINICAL CASE NUMBER: U1

BREED: Aberdeen Angus X AGE 11 yrs SEX: Female DATE OF REFERRAL: 13.2.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Ederline, Ford, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Frequent throughout year but particularly during winter.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E167 CLINICAL CASE NUMBER: U2

BREED: Aberdeen Angus X AGE 6 yrs SEX: Female DATE OF REFERRAL: 17.7.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Duodenal adenoma

FARM OF ORIGIN Bohutine, Roybridge, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Animal kept as house cow. Access June - August.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E168 CLINICAL CASE NUMBER: U3

BREED: Aberdeen Angus X AGE 5 yrs SEX: Female DATE OF REFERRAL: 4.8.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, oesophagus (> 15)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER Duodenal and colonic adenomas

FARM OF ORIGIN Glebe, Boat of Garten, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (30% of pastures)

ACCESS Frequent

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at local market

EPIDEMIOLOGY CASE NUMBER: E169 CLINICAL CASE NUMBER: U4

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 3.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (12)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Duodenal adenoma

FARM OF ORIGIN Rainton, Gatehouse, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (confirmed V.S.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E170 CLINICAL CASE NUMBER: U5

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 3.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Pharynx, cardia (4)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Rainton, Gatehouse, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (Confirmed V.S.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E171 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 11 yrs SEX: Female DATE OF REFERRAL: 13.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC Duodenum

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Barcaldine Croft, Barcaldine, Argyll

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Severe (38% of pastures)

ACCESS Continuous between 4 and 7 years of age, seldom subsequently.

INCIDENTS OF ACUTE BRACKEN POISONING Calf (Farmer).

PREVIOUS ORIGINS Kintyre, Argyll. Farm unknown

EPIDEMIOLOGY CASE NUMBER: E172 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 10.12.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (3)

IAC -

MUBN Adenocarcinoma

BUBN -

OTHER -

FARM OF ORIGIN Shielhill, Inverkip, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (9% of pastures)

ACCESS Frequent when less than 3 years of age, occasionally thereafter.

INCIDENTS OF ACUTE BRACKEN POISONING Adults

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E173 CLINICAL CASE NUMBER: U8

BREED: Aberdeen Angus AGE 10 yrs SEX: Female DATE OF REFERRAL: 27.12.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Glencalvie, Ardgay, Ross and Cromarty

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (38% of pastures)

ACCESS Continuous throughout life. Particularly when on hill grazing in summer.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults, including Case Nos. E116, E117 (confirmed self).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E174 CLINICAL CASE NUMBER: U10

BREED: Aberdeen Angus X AGE 12 yrs SEX: Female DATE OF REFERRAL: 19.2.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Oesophagus (1)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Cystademoma of thyroid, acidophil adenoma of pituitary, adenomatous hyperplasia of small intestine

FARM OF ORIGIN Low Milton, Drumnadrochit, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (58% of pastures)

ACCESS Intermittent access throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E175 CLINICAL CASE NUMBER: U11

BREED: Hereford X AGE 3 yrs SEX: Female DATE OF REFERRAL: 12.3.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Rainton, Gatehouse, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers (Confirmed v.s.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E176 CLINICAL CASE NUMBER: U13

BREED: Highland AGE 12 yrs SEX: Female DATE OF REFERRAL: 19.4.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, pharynx (6)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Inveruglas, Arrochar, Dunbartonshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E177 CLINICAL CASE NUMBER: U17

BREED: Aberdeen Angus AGE 6 yrs SEX: Female DATE OF REFERRAL: 2.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (3)

IAC -

MUBN Haemangiosarcoma

BUBN -

OTHER -

FARM OF ORIGIN Brin Mains, Stratherrick, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at > 5 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Purchased at market 3½ months prior to referral

EPIDEMIOLOGY CASE NUMBER: E178 CLINICAL CASE NUMBER: U20

BREED: Aberdeen Angus AGE 16 yrs SEX: Female DATE OF REFERRAL: 3.6.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus (6)

IAC -

MUBN Transitional cell carcinoma

BUBN Fibromas

OTHER Adrenal cortical adenoma, adenomatous hyperplasia of duodenum and colon

FARM OF ORIGIN Cairndhu, Onich, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS March - September every year.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E179 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 10 yrs SEX: Female DATE OF REFERRAL: 8.12.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus (12)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER Fibroma of oesophagus, adenomatous hyperplasia of duodenum and jejunum

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E180 CLINICAL CASE NUMBER: U22

BREED: Shorthorn X AGE 8 yrs SEX: Female DATE OF REFERRAL: 11.3.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus (9)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E181 CLINICAL CASE NUMBER: U23

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 10.1.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Morton Mains, Thornhill, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (23% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E182 CLINICAL CASE NUMBER: U26

BREED: Galloway X AGE 12 yrs SEX: Female DATE OF REFERRAL: 6.1.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus (> 10)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Bought in at one month of age

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E183 CLINICAL CASE NUMBER: U27

BREED: Luing AGE 8 yrs SEX: Female DATE OF REFERRAL: 25.1.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (3)

IAC -

MUBN Squamous cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Maam, Inveraray, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E184 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 6 yrs SEX: Female DATE OF REFERRAL: 29.11.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Oesophageal groove (3)

IAC -

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Wester Ochter Muthill, Muthill, Perthshire

HOME BRED OR BOUGHT IN Bought in at > 5 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown.

EPIDEMIOLOGY CASE NUMBER: E185 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 2.4.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (5)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION (Severe 60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E186 CLINICAL CASE NUMBER: U9

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 13.2.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, oesophagus (> 10)

IAC -

MUBN Transitional cell carcinoma

BUBN -

OTHER -

FARM OF ORIGIN Burney End, Blawith, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E187 CLINICAL CASE NUMBER: -

BREED: Hereford X Charolais AGE 10 yrs SEX: Female DATE OF REFERRAL: 31.7.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, pharynx, oesophagus, rumen (> 20)

IAC Duodenum

MUBN Transitional cell carcinoma

BUBN Haemangiomas

OTHER Adenomas of duodenum and jejunum

FARM OF ORIGIN East Waite, Wasdale, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Adults

PREVIOUS ORIGINS -

APPENDIX 4.5

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA OF ANIMALS WITH BENIGN URINARY BLADDER NEOPLASMS.

(Animals which were also affected by malignant neoplasia are recorded in Appendices 4.1 and 4.4).

EPIDEMIOLOGY CASE NUMBER: E188 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 6 yrs SEX: Female DATE OF REFERRAL: 24.4.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus (2)

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Glencalvie, Ardgay, Ross and Cromarty

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (38% of pastures)

ACCESS Continuous throughout life. Particularly when on hill grazing in summer.

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults including case Nos. E116, E117 (confirmed se

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E189 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 8 yrs SEX: Female DATE OF REFERRAL: 7.9.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate (2)

IAC -

MUBN

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Killean, Furnace, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (53% of pastures)

ACCESS Continuous during summer months. Housed in winter.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Coniirmed v.s.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E190 CLINICAL CASE NUMBER: U6

BREED: Highland X AGE 8 yrs SEX: Female DATE OF REFERRAL: 11.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (3)

IAC -

MUBN -

BUBN Haemangioma

OTHER -

FARM OF ORIGIN Grigadale, Kilchoan, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E191 CLINICAL CASE NUMBER: U12

BREED: Ayrshire AGE 7 yrs SEX: Female DATE OF REFERRAL: 11.4.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Rumen (2)

IAC -

MUBN -

BUBN Haemangiomas

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Ardchatten Home Farm, Bonawe, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (48% of pastures)

ACCESS Frequent as calf, occasional as adult.

INCIDENTS OF ACUTE BRACKEN POISONING Calves (Farmer).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E192 CLINICAL CASE NUMBER: U14

BREED: Ayrshire AGE 10 yrs SEX: Female DATE OF REFERRAL: 30.4.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN Haemangioma

OTHER -

FARM OF ORIGIN Roberton, Borgue, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Frequent as calf, seldom as adult.

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (Confirmed V.S.).

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E193 CLINICAL CASE NUMBER: -

BREED: Red Poll AGE 12 yrs SEX: Female DATE OF REFERRAL: 17.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus, cardia, oesophageal groove (> 30)

IAC -

MUBN -

BUBN Haemagnioma

OTHER Adenomatous hyperplasia of duodenium

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Bought in at one month of age

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.).

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E194 CLINICAL CASE NUMBER: -

BREED: Hereford X AGE 9 yrs SEX: Female DATE OF REFERRAL: 17.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (5)

IAC -

MUBN -

BUBN Fibromas

OTHER -

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E195 CLINICAL CASE NUMBER: U15

BREED: Hereford X AGE 11 yrs SEX: Female DATE OF REFERRAL: 12.12.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, oesophagus, rumen (7)

IAC Jejunum

MUBN -

BUBN Haemangiomas

OTHER Adenomatous hyperplasia of duodenum and jejunum

FARM OF ORIGIN Balliemeanoch, Gribun, Mull, Argyll

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Moderate (5% of pastures)

ACCESS Continuous throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.).

PREVIOUS ORIGINS Iona, Argyll

EPIDEMIOLOGY CASE NUMBER: E196 CLINICAL CASE NUMBER: U16

BREED: Friesian AGE 12 yrs SEX: Female DATE OF REFERRAL: 12.12.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Rhugarbh, Barcaldine, Argyll

HOME BRED OR BOUGHT IN Bought in at 5 years of age

BRACKEN INFESTATION Severe (40% of pastures)

ACCESS Continuous when on farm of origin.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Local farm, Barcaldine, Argyll

EPIDEMIOLOGY CASE NUMBER: E197 CLINICAL CASE NUMBER: U19

BREED: Shorthorn X AGE 8 yrs SEX: Female DATE OF REFERRAL: 19.5.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Cardia (2)

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Munigierie, Invergarry, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Continuous when on farm of origin.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Arisaig, Inverness-shire

EPIDEMIOLOGY CASE NUMBER: E198 CLINICAL CASE NUMBER: U21

BREED: Aberdeen Angus AGE 12 yrs SEX: Female DATE OF REFERRAL: 19.6.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, oesophagus, cardia (12)

IAC -

MUBN -

BUBN Haemangiomas

OTHER Papillomas of teats, adenomatous hyperplasia of duodenum and jejunum

FARM OF ORIGIN Kilfinan, Spean Bridge, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Severe (33% of pastures)

ACCESS Frequent. Attempt made to keep cattle off bracken-infested pastures when greater risk of acute bracken poisoning.

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (Farmer).

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E199 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 8 yrs SEX: Female DATE OF REFERRAL: 8.12.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue (1)

IAC -

MUBN -

BUBN Fibromas

OTHER Adenomatous hyperplasia of small intestine

FARM OF ORIGIN Barrahormaid, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E200 CLINICAL CASE NUMBER: U24

BREED: Shorthorn X AGE 11 yrs SEX: Female DATE OF REFERRAL: 25.1.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue (1)

IAC -

MUBN -

BUBN Haemangioma

OTHER -

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E201 CLINICAL CASE NUMBER: U25

BREED: Shorthorn X AGE 9 yrs SEX: Female DATE OF REFERRAL: 20.8.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Lower Gartally, Drumnadrochit, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Confirmed throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E202 CLINICAL CASE NUMBER: -

BREED: Galloway X AGE 9 yrs SEX: Female DATE OF REFERRAL: 17.2.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN Fibromas

OTHER -

FARM OF ORIGIN Bennon, Tynron, Thornhill, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 9 months of age

BRACKEN INFESTATION Severe (23% of pastures)

ACCESS Continuous when on farm of origin.

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s.).

PREVIOUS ORIGINS Purchased at Newcastleton market

EPIDEMIOLOGY CASE NUMBER: E203 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 14 yrs SEX: Female DATE OF REFERRAL: 2.4.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (7)

IAC -

MUBN -

BUBN Haemangiomas

OTHER -

FARM OF ORIGIN Gallachoille, Tayvallich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E204 CLINICAL CASE NUMBER: -

BREED: Hereford AGE 7 yrs SEX: Female DATE OF REFERRAL: 20.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, oesophagus, rumen (> 20)

IAC -

MUBN -

BUBN Haemangioma, fibromas

OTHER Adenomatous hyperplasia of duodenum

FARM OF ORIGIN Christcliffe, Boot, Eskdale, Cumbria

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous when on farm of origin.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Local farm, Eskdale, Cumbria

EPIDEMIOLOGY CASE NUMBER: E205 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE Unknown SEX: Female DATE OF REFERRAL: 24.4.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN Haemangioma

OTHER -

FARM OF ORIGIN Little Mark, New Cumnock, Ayrshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

APPENDIX 4.6

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA OF ADULT ANIMALS WITH OTHER MALIGNANT NEOPLASMS.

EPIDEMIOLOGY CASE NUMBER: E206 CLINICAL CASE NUMBER: M25

BREED: Friesian AGE 4 yrs SEX: Female DATE OF REFERRAL: 28.9.71

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN East Mitchelton, Lochwinnoch, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E207 CLINICAL CASE NUMBER: M27

BREED: Ayrshire AGE5 yrs SEX:Female DATE OF REFERRAL: 30.5.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Ramageton, Mauchline, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E208 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 10 yrs SEX: Female DATE OF REFERRAL: 2.8.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Anaplastic Sarcoma (No primary site identified)

FARM OF ORIGIN West Carswell, Neilston, Renfrewshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E209 CLINICAL CASE NUMBER: -

BREED: Charolais AGE 3 yrs SEX: Female DATE OF REFERRAL: 20.8.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Friorton, Newport, Fife

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E210 CLINICAL CASE NUMBER: -

BREED: Friesian AGE 4 yrs SEX:Female DATE OF REFERRAL: 12.11.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Bronchial carcinoma

FARM OF ORIGIN Croftfoot, Catrine, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E211 CLINICAL CASE NUMBER: AL3

BREED: Hereford X AGE 12 yrs SEX: Female DATE OF REFERRAL: 10.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Nether Largie, Kilmartin, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (70% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E212 CLINICAL CASE NUMBER: -

BREED: Hereford X AGE 5 yrs SEX: Female DATE OF REFERRAL: 26.3.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Rumen (2)

IAC -

MUBN -

BUBN -

OTHER Ocular squamous cell carcinoma

FARM OF ORIGIN Dunain Mains, Inverness, Inverness-shire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (10% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E213 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 10 yrs SEX: Female DATE OF REFERRAL: 24.5.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Squamous carcinoma of small intestine

FARM OF ORIGIN Bailliemeanoch, Cladich, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (42% of pastures)

ACCESS Continous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E214 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 1.6.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Ovarian carcinoma

FARM OF ORIGIN South Brownhills, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E215 CLINICAL CASE NUMBER: -

BREED: Galloway AGE > 10 yrs SEX: Female DATE OF REFERRAL: 5.6.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, pharynx (> 10)

IAC -

MUBN -

BUBN -

OTHER Malignant melanoma

FARM OF ORIGIN Raehills, St. Annes, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (16% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E216 CLINICAL CASE NUMBER: AL1

BREED: Friesian AGE 4 yrs SEX: Female DATE OF REFERRAL: 29.6.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Balure Shian, Benderloch, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (1% of pastures)

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E217 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 4 yrs SEX: Female DATE OF REFERRAL: 29.9.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Granulosa cell tumour

FARM OF ORIGIN Turnberry Lodge , Turnberry, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E218 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 11 yrs SEX: Female DATE OF REFERRAL: 4.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, oesophagus (8)

IAC -

MUBN -

BUBN -

OTHER Squamous cell carcinoma (No primary site identified)

FARM OF ORIGIN Accuraich, Cladich, Argyll

HOME BRED OR BOUGHT IN Bought in at 6 months of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Dalmally market.

EPIDEMIOLOGY CASE NUMBER: E219 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 10.10.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Peritoneal Fibrosarcoma

FARM OF ORIGIN East Hook Head, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E220 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 12 yrs SEX: Female DATE OF REFERRAL: 4.11.73

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Pharynx, oesophagus, rumen (11)

IAC -

MUBN -

BUBN -

OTHER Thyroid carcinoma

FARM OF ORIGIN Drum, Kilfinan, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (13% of pasture)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Heifers, adults (confirmed v.s. and self)

PREVIOUS ORIGINS Unknown. Purchased at Strachur Sale.

EPIDEMIOLOGY CASE NUMBER: E221 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 9 yrs SEX: Female DATE OF REFERRAL: 28.5.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Pharynx (6)

IAC -

MUBN -

BUBN -

OTHER Osteosarcoma of pelvis, haemangiomas of nasal epithelium

FARM OF ORIGIN Crichen, Moniave, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E222 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 10.9.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Cholangiocarcinoma

FARM OF ORIGIN South Veterhill, Dunlop, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased from cattle dealer.

EPIDEMIOLOGY CASE NUMBER: E223 CLINICAL CASE NUMBER: -

BREED: Luing AGE 11 yrs SEX: Female DATE OF REFERRAL: 3.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Bronchial carcinoma

FARM OF ORIGIN Ederline, Ford, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Freuqne throughout year but particularly during winter

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E224 CLINICAL CASE NUMBER: M26

BREED: Friesian AGE 5 yrs SEX: Female DATE OF REFERRAL: 5.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Keld, Kings Meaburn, Penrith, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E225 CLINICAL CASE NUMBER: -

BREED: Galloway X AGE 16 yrs SEX: Female DATE OF REFERRAL: 17.10.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Oesophagus (16)

IAC -

MUBN -

BUBN -

OTHER Renal haemangiosarcoma

FARM OF ORIGIN Brenfield, Ardrishaig, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (20% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E226 CLINICAL CASE NUMBER: AL2

BREED: Friesian X AGE 6 yrs SEX: Female DATE OF REFERRAL: 13.11.74

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Gartverrie, Glenboig, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E227 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 13 yrs SEX: Female DATE OF REFERRAL: 13.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Anaplastic uterine carcinoma

FARM OF ORIGIN Rowallan Home Farm, Fenwick, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E228 CLINICAL CASE NUMBER: -

BREED: Hereford X AGE 8 yrs SEX: Female DATE OF REFERRAL: 24.3.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (2)

IAC -

MUBN -

BUBN -

OTHER Ocular squamous cell carcinoma

FARM OF ORIGIN Gorteneorn, Acharacle, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Moderate (17% of pastures)

ACCESS Frequent, particularly during summer months

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Langal, Acharacle, Argyll. Bracken infested

EPIDEMIOLOGY CASE NUMBER: E229 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 5 yrs SEX: Female DATE OF REFERRAL: 19.4.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Bronchial carcinoma

FARM OF ORIGIN Brock Loch, Kirkpatrick Durham, Kirkcudbright.

HOME BRED OR BOUGHT IN Bought in at one year of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Frequent throughout adult life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Castle Douglas Market.

EPIDEMIOLOGY CASE NUMBER: E230 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 14 yrs SEX: Female DATE OF REFERRAL: 26.11.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Cholangiocarcinoma

FARM OF ORIGIN Abbey, Crieff, Perthshire

HOME BRED OR BOUGHT IN Bought in at one year of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown.

EPIDEMIOLOGY CASE NUMBER: E231 CLINICAL CASE NUMBER: -

BREED: Friesian AGE 7 yrs SEX: Female DATE OF REFERRAL: 2.3.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Haemangiosarcoma of skin at base of horn

FARM OF ORIGIN Drums, Langbank, Renfrewshire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Light (1% of pastures)

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Mayne, Elgin. Free from bracken infestation.

EPIDEMIOLOGY CASE NUMBER: E232 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 9 yrs SEX: Female DATE OF REFERRAL: 16.4.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate, pharynx (12)

IAC -

MUBN -

BUBN -

OTHER Splenic sarcoma

FARM OF ORIGIN Heather Cottage, Faynuilt, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS April to October every year as adult

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Uist, Outer Hebrides. Bracken status unknown.

EPIDEMIOLOGY CASE NUMBER: E233 CLINICAL CASE NUMBER: M28

BREED: Friesian AGE 11 yrs SEX: Female DATE OF REFERRAL: 12.1077

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Lymphosarcoma

FARM OF ORIGIN Laigh Mains, Thornhill, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E234 CLINICAL CASE NUMBER: -

BREED: Galloway AGE 15 yrs SEX: Female DATE OF REFERRAL: 6.4.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (1)

IAC -

MUBN -

BUBN -

OTHER Cholangiocarcinoma

FARM OF ORIGIN Blindhillbush, Lockerbie, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 8 years of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adult (Farmer)

PREVIOUS ORIGINS Kilquhockadale, Kirkcowan, Wigtownshire. Bracken status unknown.

EPIDEMIOLOGY CASE NUMBER: E235 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 11 yrs SEX: Female DATE OF REFERRAL: 8.11.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Adrenal carcinoma

FARM OF ORIGIN Savary, Morven, Argyll

HOME BRED OR BOUGHT IN Bought in at 8 years of age

BRACKEN INFESTATION Severe (56% of pastures)

ACCESS Continuous when adult

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Oban, Argyll area. Exact farm unknown.

EPIDEMIOLOGY CASE NUMBER: E236 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 8 yrs SEX: Female DATE OF REFERRAL: 21.11.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Palate (3)

IAC -

MUBN -

BUBN -

OTHER Thyroid carcinoma

FARM OF ORIGIN Dawcrue, Methven, Perthshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Frequent but only as an adult

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E237 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 9 yrs SEX: Female DATE OF REFERRAL: 13.12.78

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma of Mammary Gland

FARM OF ORIGIN Ardfenaig, Bunessan, Mull, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Calves, heifers, adults (confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E238 CLINICAL CASE NUMBER: -

BREED: Hereford AGE 8 yrs SEX:Female DATE OF REFERRAL: 10.1.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Uterine carcinoma

FARM OF ORIGIN Hillside, Kilmacolm, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E239 CLINICAL CASE NUMBER: -

BREED: Friesian AGE 3 yrs SEX: Female DATE OF REFERRAL: 19.2.71

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Malignant ovarian thecoma

FARM OF ORIGIN High Brankle, Bridge of Weir, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E240 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 7 yrs SEX: Female DATE OF REFERRAL: 20.2.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Cholangiocarcinoma

FARM OF ORIGIN Newfield Mains, Dundonald, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown, purchased from cattle dealer.

EPIDEMIOLOGY CASE NUMBER: E241 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus X AGE 15 yrs SEX: Female DATE OF REFERRAL: 3.4.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma (Primary site not identified)

FARM OF ORIGIN Gallanoch Home Farm, Oban, Argyll

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Severe (25% of pastures)

ACCESS Occasionally when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Heifers (Farmer)

PREVIOUS ORIGINS Exact farm unknown. Purchased through dealer from Islay.

EPIDEMIOLOGY CASE NUMBER: E242 CLINICAL CASE NUMBER: -

BREED: Hereford AGE 9 yrs SEX: Female DATE OF REFERRAL: 18.5.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Ocular squamous cell carcinoma

FARM OF ORIGIN Finzean, Banchory , Aberdeenshire.

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Moderate (8% of pastures)

ACCESS Occasionally when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Ireland

EPIDEMIOLOGY CASE NUMBER: E243 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 11 yrs SEX: Female DATE OF REFERRAL: 28.5.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Ocular Squamous cell carcinoma

FARM OF ORIGIN Corranmore, Ardfern, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E244 CLINICAL CASE NUMBER: -

BREED: Shorthorn AGE 7 yrs SEX: Female DATE OF REFERRAL: 30.5.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma of mandible

FARM OF ORIGIN Blar Macfoldach, Fort William, Inverness-shire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Moderate (13% of pastures)

ACCESS Frequent when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Glen Nevis Farms, Fort William. Bracken infested.

EPIDEMIOLOGY CASE NUMBER: E245 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 7 yrs SEX: Female DATE OF REFERRAL: 1.6.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma of forelimb

FARM OF ORIGIN Turnberry Lodge, Turnberry, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E246 CLINICAL CASE NUMBER: -

BREED: Jersey AGE 10 yrs SEX: Female DATE OF REFERRAL: 2.12.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Ovarian carcinoma

FARM OF ORIGIN Canada Farm, Moresby, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E247 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE Aged SEX: Female DATE OF REFERRAL: 7.11.72

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Mammary carcinoma

FARM OF ORIGIN Blackstone, Dalry, Ayrshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E248 CLINICAL CASE NUMBER: -

BREED: Shorthorn X AGE 10 yrs SEX: Female DATE OF REFERRAL: 31.774

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Tongue, palate, oesophagus (> 20)

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma of mandible

FARM OF ORIGIN Blairmore, Dunoon, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E249 CLINICAL CASE NUMBER: -

BREED: Luing AGE Aged SEX: Female DATE OF REFERRAL: 11.5.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Anaplastic carcinoma (No primary site identified)

FARM OF ORIGIN Kilninver, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E250 CLINICAL CASE NUMBER: -

BREED: Aberdeen Angus AGE 13 yrs SEX: Female DATE OF REFERRAL: 23.6.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Cholangiocarcinoma

FARM OF ORIGIN Altna Baben, Spean Bridge, Inverness-shire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E251 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 5 yrs SEX: Female DATE OF REFERRAL: 12.12.75

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Adenocarcinoma of lung

FARM OF ORIGIN Drumore, Campbelltown, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E252 CLINICAL CASE NUMBER: -

BREED: Hereford AGE 6 yrs SEX: Female DATE OF REFERRAL: 3.6.76

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Adenocarcinoma of uterus

FARM OF ORIGIN Birkhill, Coalburn, Lanarkshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E253 CLINICAL CASE NUMBER: -

BREED: Hereford AGE 5 yrs SEX: Female DATE OF REFERRAL: 7.6.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Bronchial carcinoma

FARM OF ORIGIN High Smithstone, Lochwinnoch, Argyll

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E254 CLINICAL CASE NUMBER: -

BREED: Ayrshire AGE 6 yrs. SEX: Female DATE OF REFERRAL: 4.11.77

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP Rumen (1)

IAC -

MUBN -

BUBN -

OTHER Fibrosarcoma of Mandible

FARM OF ORIGIN Fowler, Mauchline, Ayrshire

HOME BRED OR BOUGHT IN NA

BRACKEN INFESTATION NA

ACCESS NA

INCIDENTS OF ACUTE BRACKEN POISONING NA

PREVIOUS ORIGINS NA

EPIDEMIOLOGY CASE NUMBER: E255 CLINICAL CASE NUMBER: -

BREED: Hereford X AGE 10 yrs SEX: Female DATE OF REFERRAL: 28.1.79

NEOPLASMS IDENTIFIED (SITE, NUMBER AND TYPE WHERE RELEVANT)

UASCC -

UAP -

IAC -

MUBN -

BUBN -

OTHER Uterine Carcinoma

FARM OF ORIGIN Dunmore, Seil, Oban, Argyll

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Farmer)

PREVIOUS ORIGINS Achnalarig, Oban, Argyll. Bracken infested.

APPENDIX 4.7

EPIDEMIOLOGICAL AND PATHOLOGICAL DATA

OF CONTROL ANIMALS WITHOUT NEOPLASIA.

EPIDEMIOLOGY CASE NUMBER: E256

BREED: Highland AGE: 7 yrs SEX: Female DATE OF REFERENCE: 7.4.76

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis

FARM OF ORIGIN Summer Hill, Campbeltown, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (12% of pastures)

ACCESS Frequent throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E257

BREED: Galloway AGE: 14 yrs SEX: Female DATE OF REFERENCE: 10.4.76

CAUSE OF DEATH/SLAUGHTER Dryopteris felix mas poisoning

FARM OF ORIGIN Traquhair, Balmaclellan, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E258

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 13.4.76

CAUSE OF DEATH/SLAUGHTER: Diffuse Fibrosing Aeveolitis

FARM OF ORIGIN Whingill, Hartley, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E259

BREED: Shorthorn AGE: 10 yrs SEX: Female DATE OF REFERENCE: 14.4.76

CAUSE OF DEATH/SLAUGHTER Pharyngeal cellulitis

FARM OF ORIGIN Townfoot, Thornhill, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Light (1% of pastures). Confined to hill grazing

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Ireland. Bracken exposure unknown.

EPIDEMIOLOGY CASE NUMBER: E260

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 1.5.76

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN Townend, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E261

BREED: Ayrshire AGE: 10 yrs SEX: Female DATE OF REFERENCE: 10.5.76

CAUSE OF DEATH/SLAUGHTER Deformed tracheal cartilages

FARM OF ORIGIN Valleyfield, Rait, Perthshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Cluniemore, Pitlochry, Perthshire.
Bracken status unknown.

EPIDEMIOLOGY CASE NUMBER: E262

BREED: Friesian AGE: 8 yrs SEX: Female DATE OF REFERENCE: 11.5.76

CAUSE OF DEATH/SLAUGHTER: Haemolytic anaemia of unknown origin

FARM OF ORIGIN Devol, Port Glasgow, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E263

BREED: Aberdeen Angus AGE: 8 yrs SEX: Female DATE OF REFERENCE: 22.5.76

CAUSE OF DEATH/SLAUGHTER Salmonellosis

FARM OF ORIGIN Lagalgarve, Campbeltown, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Only occasional access when over 3 years of age

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E264

BREED: Aberdeen Angus AGE: 4 yrs SEX: Female DATE OF REFERENCE: 7.6.76

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN Airdsgreen, Glenbuck, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Moderate (7% of pastures)

ACCESS Occassional

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E265

BREED: Ayrshire AGE: 4 yrs SEX: Female DATE OF REFERENCE: 16.6.76

CAUSE OF DEATH/SLAUGHTER Endocarditis

FARM OF ORIGIN Roddinghill, Irvine, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E266

BREED: Aberdeen Angus X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 5.8.76

CAUSE OF DEATH/SLAUGHTER: Abscess of oral/nasal passages

FARM OF ORIGIN: Quarry, Sedburgh, Cumbria

HOME BRED OR BOUGHT IN: Bought in at 6 years of age

BRACKEN INFESTATION: Nil

ACCESS: Never

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown

EPIDEMIOLOGY CASE NUMBER: E267

BREED: Ayrshire AGE: 7 yrs SEX: Female DATE OF REFERENCE: 25.9.76

CAUSE OF DEATH/SLAUGHTER: Chronic suppurative pneumonia

FARM OF ORIGIN: Dalswinton, Dumfries, Dumfriesshire

HOME BRED OR BOUGHT IN: Home bred

BRACKEN INFESTATION: Nil

ACCESS: Never

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: -

EPIDEMIOLOGY CASE NUMBER: E268

BREED: Friesian AGE: 4 yrs SEX: Female DATE OF REFERENCE: 2.10.76

CAUSE OF DEATH/SLAUGHTER: Ragwort Poisoning

FARM OF ORIGIN Maryport, Stranraer, Wigtownshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E269

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 8.10.76

CAUSE OF DEATH/SLAUGHTER Mastitis, mammary vein thrombosis

FARM OF ORIGIN Snade Mill, Dunscore, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Rare, only as a calf and heifer.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E270

BREED: Luing AGE: 5 yrs SEX: Female DATE OF REFERENCE: 19.11.76

CAUSE OF DEATH/SLAUGHTER: Urolithiasis, pyelonephritis

FARM OF ORIGIN: Strone, Minard, Argyll

HOME BRED OR BOUGHT IN: Bought in at 2 years of age

BRACKEN INFESTATION: Light (4% of pastures)

ACCESS: Occasional as adult. Continuous on previous farm of origin. (See below).

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Caddleton, Balvicar, Oban. Severe bracken infestation (See Case No. E81)

EPIDEMIOLOGY CASE NUMBER: E271

BREED: ShorthornX AGE: 10 yrs SEX: Female DATE OF REFERENCE: 23.11.76

CAUSE OF DEATH/SLAUGHTER: Johnes Disease

FARM OF ORIGIN: Harvies Mailing, Denny, Stirlingshire

HOME BRED OR BOUGHT IN: Bought in at 6 years of age

BRACKEN INFESTATION: Light (< 1% of pastures)

ACCESS: Rare

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown

EPIDEMIOLOGY CASE NUMBER: E272

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 6.1.77

CAUSE OF DEATH/SLAUGHTER: Posterior vena caval thrombosis with embolic pneumonia

FARM OF ORIGIN M'Nairson, Ayr, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasional throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E273

BREED: Hereford AGE: 7 yrs SEX: Female DATE OF REFERENCE: 19.1.77

CAUSE OF DEATH/SLAUGHTER Abomasal ulceration, omasitis

FARM OF ORIGIN Newmains, Inchinnan, Remfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E274

BREED: Friesian AGE: 9 yrs SEX: Female DATE OF REFERENCE: 1.2.77

CAUSE OF DEATH/SLAUGHTER: Hydrops allantois

FARM OF ORIGIN Lampits, Carstairs, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E275

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 9.2.77

CAUSE OF DEATH/SLAUGHTER Renal amyloidosis

FARM OF ORIGIN Gotterbie, Lochmaben, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E276

BREED: Shorthorn AGE: 8 yrs SEX: Female DATE OF REFERENCE: 25.2.77

CAUSE OF DEATH/SLAUGHTER: Johnes disease

FARM OF ORIGIN Beattock, Moffat, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS

EPIDEMIOLOGY CASE NUMBER: E277

BREED: Galloway AGE: 9 yrs SEX: Female DATE OF REFERENCE: 1.3.77

CAUSE OF DEATH/SLAUGHTER Chronic suppernative pneumonia

FARM OF ORIGIN Glenhead, Girvan, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E278

BREED: Ayrshire AGE: 7 yrs SEX: Female DATE OF REFERENCE: 11.3.77

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN Starbins, Lesmahgon, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E279

BREED: Hereford AGE: 11 yrs SEX: Female DATE OF REFERENCE: 20.4.77

CAUSE OF DEATH/SLAUGHTER Metritis, abdominal abscessation

FARM OF ORIGIN Spango, Crawfordjohn, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Occasional

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E280

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 29.4.77

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis

FARM OF ORIGIN Raahead, East Kilbride, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E281

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 18.5.77

CAUSE OF DEATH/SLAUGHTER Abomasitis

FARM OF ORIGIN Monkton Hill, Monkton, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E282

BREED: Hereford X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 24.5.77

CAUSE OF DEATH/SLAUGHTER: Intussusception

FARM OF ORIGIN Culgruff, Crossmichael, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E283

BREED: Ayrshire AGE: 8 yrs SEX: Female DATE OF REFERENCE: 1.6.77

CAUSE OF DEATH/SLAUGHTER Diffuse Fibrosing Alveolitis

FARM OF ORIGIN West Mitchelton, Lochwinnoch, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasional

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E284

BREED: Aberdeen Angus X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 2.6.77

CAUSE OF DEATH/SLAUGHTER: Diffuse Fibrosing Alveolitis

FARM OF ORIGIN: Chapelton Mains, Stewarton, Ayrshire

HOME BRED OR BOUGHT IN: Bought in at 5 years of age

BRACKEN INFESTATION: Nil

ACCESS: Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Argyll. Exact farm and bracken status unknown

EPIDEMIOLOGY CASE NUMBER: E285

BREED: Shorthorn X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 6.6.77

CAUSE OF DEATH/SLAUGHTER: Ostertagiasis abomasal ulceration, peritoni

FARM OF ORIGIN: Corramore, Kirkfieldbank, Lanarkshire

HOME BRED OR BOUGHT IN: Bought in at 5 years of age

BRACKEN INFESTATION: Nil

ACCESS: Never

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown

EPIDEMIOLOGY CASE NUMBER: E286

BREED: Aberdeen Angus X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 11.6.77

CAUSE OF DEATH/SLAUGHTER: Johnes disease

FARM OF ORIGIN Southend, Campbeltown, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (40% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E287

BREED: Shorthorn X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 14.6.77

CAUSE OF DEATH/SLAUGHTER Johnes disease

FARM OF ORIGIN South Crubasdale, Campbeltown, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Frequent as a calf/heifer.
No access when adult.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E288

BREED: South Avon AGE: 6 yrs SEX: Female DATE OF REFERENCE: 18.7.77

CAUSE OF DEATH/SLAUGHTER: Chronic suppurative pneumonia

FARM OF ORIGIN Cassafuir, Port of Mentieth, Stirlingshire

HOME BRED OR BOUGHT IN Homebred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasionally during summer. Also in winter if outwintered.

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E289

BREED: Galloway AGE: 10 yrs SEX: Female DATE OF REFERENCE: 2.9.77

CAUSE OF DEATH/SLAUGHTER Chronic interstitial pneumonia, cor pulmonale

FARM OF ORIGIN Linfairin, Straiton, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Frequent throughout life.

INCIDENTS OF ACUTE BRACKEN POISONING 2 Year old bullock (Farmer)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E290

BREED: Ayrshire AGE: 4 yrs SEX: Female DATE OF REFERENCE: 3.9.77

CAUSE OF DEATH/SLAUGHTER: Endocarditis

FARM OF ORIGIN: Gateside, Ayr, Ayrshire

HOME BRED OR BOUGHT IN: Home bred

BRACKEN INFESTATION: Nil

ACCESS: Never

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: -

EPIDEMIOLOGY CASE NUMBER: E291

BREED: Galloway AGE: 4 yrs SEX: Female DATE OF REFERENCE: 8.9.77

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis

FARM OF ORIGIN: Auchenhessnane, Penpont, Dumfriesshire

HOME BRED OR BOUGHT IN: Bought in at one week of age

BRACKEN INFESTATION: Severe (> 20% of pastures)

ACCESS: Continuous throughout life

INCIDENTS OF ACUTE BRACKEN POISONING: Heifers, adults (Confirmed v.s.)

PREVIOUS ORIGINS: Unknown

EPIDEMIOLOGY CASE NUMBER: E292

BREED: Ayrshire AGE: 8 yrs SEX: Female DATE OF REFERENCE: 27.10.77

CAUSE OF DEATH/SLAUGHTER: Endocarditis

FARM OF ORIGIN Humestone, Maybole, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasional, only as a heifer

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E293

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 2.11.77

CAUSE OF DEATH/SLAUGHTER Traumatic pericarditis

FARM OF ORIGIN Greenshields, Carnwath, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E294

BREED: Shorthorn X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 14.11.77

CAUSE OF DEATH/SLAUGHTER: No abnormality detected

FARM OF ORIGIN Kenmuir, New Galloway, Kirkcudbright

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Rare

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E295

BREED: Friesian AGE: 4 yrs SEX: Female DATE OF REFERENCE: 23.12.77

CAUSE OF DEATH/SLAUGHTER Posterior vena caval thrombosis with embolic pneumonia

FARM OF ORIGIN Fullshawood, Ayr, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E296

BREED: Friesian AGE: 9 yrs SEX: Female DATE OF REFERENCE: 6.1.78

CAUSE OF DEATH/SLAUGHTER: Endocarditis

FARM OF ORIGIN Clonherb, Fenwick, Aryshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E297

BREED: Shorthorn AGE: 9 yrs SEX: Female DATE OF REFERENCE: 13.1.78

CAUSE OF DEATH/SLAUGHTER No abnormality detected

FARM OF ORIGIN Kilmun, Dalavich, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Occasional

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Isle of Jura, Argyll
Farm bracken infested

EPIDEMIOLOGY CASE NUMBER: E298

BREED: Galloway AGE: 6 yrs SEX: Female DATE OF REFERENCE: 1.2.78

CAUSE OF DEATH/SLAUGHTER: Multiple lymph node abscessation

FARM OF ORIGIN Dalreoch Mains, Colmonell, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (60% of pastures)

ACCESS Continuous throughout life except December-April each year

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s.)

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E299

BREED: Shorthorn AGE: 8 yrs SEX: Female DATE OF REFERENCE: 8.2.78

CAUSE OF DEATH/SLAUGHTER Facial cellulitis

FARM OF ORIGIN Easton, Dunsyre, Carnwath, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 6 months of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E300

BREED: Aberdeen Angus X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 10.3.78

CAUSE OF DEATH/SLAUGHTER: Oesophagial dilitation

FARM OF ORIGIN Middleton, Comrie, Perthshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E301

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 28.3.78

CAUSE OF DEATH/SLAUGHTER Necrotising bronchopneumonia

FARM OF ORIGIN West Dykes, Drumclog, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E302

BREED: Ayrshire AGE: 9 yrs SEX: Female DATE OF REFERENCE: 10.4.78

CAUSE OF DEATH/SLAUGHTER: Chronic suppurative pneumonia

FARM OF ORIGIN Craig, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 7 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Lanark market

EPIDEMIOLOGY CASE NUMBER: E303

BREED: Hereford X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 19.4.78

CAUSE OF DEATH/SLAUGHTER Johnes disease

FARM OF ORIGIN Dunmore, Moffat, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Continuous when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E304

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 4.5.78

CAUSE OF DEATH/SLAUGHTER: Metritis

FARM OF ORIGIN Wester Boreland, Thornhill, Stirlingshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E305

BREED: Shorthorn AGE: 8 yrs SEX: Female DATE OF REFERENCE: 26.5.78

CAUSE OF DEATH/SLAUGHTER Perforation of large intestine

FARM OF ORIGIN South Uist, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E306

BREED: Ayrshire AGE: 6 yrs SEX: Female DATE OF REFERENCE: 8.6.78

CAUSE OF DEATH/SLAUGHTER: Endocarditis

FARM OF ORIGIN North Bamkhead, Avonbridge, Stirlingshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E307

BREED: Shorthorn X AGE: 8 yrs SEX: Female DATE OF REFERENCE: 13.6.78

CAUSE OF DEATH/SLAUGHTER No abnormality detected

FARM OF ORIGIN Daltot, Achnamara, Argyll

HOME BRED OR BOUGHT IN Bought in at 2 years of age

BRACKEN INFESTATION Light (2% of pastures)

ACCESS Rare, only as a heifer

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at Oban market

EPIDEMIOLOGY CASE NUMBER: E308

BREED: Ayrshire AGE: 13 yrs SEX: Female DATE OF REFERENCE: 22.6.78

CAUSE OF DEATH/SLAUGHTER: Renal amyloidosis

FARM OF ORIGIN West Mossgeil, Mauchline, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E309

BREED: Aberdeen Angus X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 15.8.78

CAUSE OF DEATH/SLAUGHTER Pyelonephritis

FARM OF ORIGIN Corran, Clachan, Argyll

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Moderate (9% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s.)

PREVIOUS ORIGINS Gartnagrenach, Whitehouse, Argyll and prior to 18 months Lergiechonie, Ardfern, Argyll

EPIDEMIOLOGY CASE NUMBER: E310

BREED: Ayrshire AGE: 6 yrs SEX: Female DATE OF REFERENCE: 30.8.78

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis and fat necrosis

FARM OF ORIGIN: Mid Brackenridge, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN: Home bred

BRACKEN INFESTATION: Nil

ACCESS: Never

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: -

EPIDEMIOLOGY CASE NUMBER: E311

BREED: Ayrshire AGE: 9 yrs SEX: Female DATE OF REFERENCE: 22.9.78

CAUSE OF DEATH/SLAUGHTER: Diffuse fibrosing alveolitis

FARM OF ORIGIN: Torfoot, Drumclog, Lanarkshire

HOME BRED OR BOUGHT IN: Bought in at 4 years of age

BRACKEN INFESTATION: Nil

ACCESS: Never on farm of origin or previous origins

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Born on owners father's farm in Northern Ireland which is bracken free

EPIDEMIOLOGY CASE NUMBER: E312

BREED: Shorthorn AGE: 9 yrs SEX: Female DATE OF REFERENCE: 5.10.78

CAUSE OF DEATH/SLAUGHTER: Diffuse fibrosing alyeolitis

FARM OF ORIGIN Mid Heilar, Sorn, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E313

BREED: Hereford AGE: 11 yrs SEX: Female DATE OF REFERENCE: 26.10.78

CAUSE OF DEATH/SLAUGHTER Infections bovine rhinotracheitis

FARM OF ORIGIN Knoweside, Maybole, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 18 months of age

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Frequent throughout life

INCIDENTS OF ACUTE BRACKEN POISONING Adults (Confirmed v.s.)

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E314

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 26.10.78

CAUSE OF DEATH/SLAUGHTER: Necrotising bronchopneumonia

FARM OF ORIGIN Saughtrees, Whamprey, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E315

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 7.11.78

CAUSE OF DEATH/SLAUGHTER Haemorrhagic diathesis

FARM OF ORIGIN Moat Mains, Lesmahagon, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E316

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 10.11.78

CAUSE OF DEATH/SLAUGHTER: Leptospirosis

FARM OF ORIGIN Gainerhill, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E317

BREED: Hereford AGE: 6 yrs SEX: Female DATE OF REFERENCE: 15.11.78

CAUSE OF DEATH/SLAUGHTER Pulmonary abscesses

FARM OF ORIGIN Laigh Carnduff, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E318

BREED: Galloway AGE: 11 yrs SEX: Female DATE OF REFERENCE: 17.11.78

CAUSE OF DEATH/SLAUGHTER: Diffuse fibrosing alveolitis

FARM OF ORIGIN Moorhouse, Orton, Cumbria

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E319

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 23.11.78

CAUSE OF DEATH/SLAUGHTER Fat necrosis

FARM OF ORIGIN Townhead, Mauchline, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E320

BREED: Aberdeen Angus AGE: 3 yrs SEX: Female DATE OF REFERENCE: 19.12.78

CAUSE OF DEATH/SLAUGHTER: Listeriosis

FARM OF ORIGIN: St. Johns Kirk, Symington, Ayrshire

HOME BRED OR BOUGHT IN: Bought in at 2 years of age

BRACKEN INFESTATION: Light (< 1% of pastures)

ACCESS: Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown. Ireland

EPIDEMIOLOGY CASE NUMBER: E321

BREED: Hereford X AGE: 5 yrs SEX: Female DATE OF REFERENCE: 11.1.79

CAUSE OF DEATH/SLAUGHTER: Cerebro-cortical necrosis

FARM OF ORIGIN: Goosehill, Sanquhar, Dumfriesshire

HOME BRED OR BOUGHT IN: Bought in at 2 weeks of age

BRACKEN INFESTATION: Moderate (7% of pastures)

ACCESS: Occasional, only as a heifer

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown

EPIDEMIOLOGY CASE NUMBER: E322

BREED: Hereford AGE: 6 yrs SEX: Female DATE OF REFERENCE: 22.1.79

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN West Glespin, Douglas, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E323

BREED: Ayrshire AGE: 10 yrs SEX: Female DATE OF REFERENCE: 1.2.79

CAUSE OF DEATH/SLAUGHTER Pyothorax

FARM OF ORIGIN Dalswinton, Auldgirth, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E324

BREED: Aberdeen Angus X AGE: 10 yrs SEX: Female DATE OF REFERENCE: 6.2.79

CAUSE OF DEATH/SLAUGHTER: Diffuse Fibrosing alveolitis

FARM OF ORIGIN Wester Mosshat, Auchengray,Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E325

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 1.3.79

CAUSE OF DEATH/SLAUGHTER Aspergillus mastitis and cervical cysts

FARM OF ORIGIN Ladehead, Lesmahagon,Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 6 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E326

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 6.3.79

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN Nether Affleck, Kirkfieldbank, Dumfriesshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E327

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 23.3.79

CAUSE OF DEATH/SLAUGHTER Traumatic pericarditis

FARM OF ORIGIN West Mains, Glassford, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E328

BREED: Friesian AGE: 4 yrs SEX: Female DATE OF REFERENCE: 13.4.79

CAUSE OF DEATH/SLAUGHTER: Traumatic pericarditis

FARM OF ORIGIN Walesley, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Homebred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E329

BREED: Shorthorn X AGE: 12 yrs SEX: Female DATE OF REFERENCE: 11.5.79

CAUSE OF DEATH/SLAUGHTER Ragwort Poisoning

FARM OF ORIGIN Crosshill, Slammanan, Stirlingshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E330

BREED: Friesian AGE: 7 yrs SEX: Female DATE OF REFERENCE: 16.5.79

CAUSE OF DEATH/SLAUGHTER: Mastitis, metritis, septicaemia

FARM OF ORIGIN Auldhouse, Renfrewshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E331

BREED: Friesian AGE: 3 yrs SEX: Female DATE OF REFERENCE: 18.5.79

CAUSE OF DEATH/SLAUGHTER Pyelonephritis

FARM OF ORIGIN Easter Bedcow, Kirkintilloch, Dumbartonshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E332

BREED: Ayrshire AGE: 11 yrs SEX: Female DATE OF REFERENCE: 24.5.79

CAUSE OF DEATH/SLAUGHTER: Renal amyloidosis

FARM OF ORIGIN Weitshaw, Sorn, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 9 years of age

BRACKEN INFESTATION Nil

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E333

BREED: Hereford X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 25.5.79

CAUSE OF DEATH/SLAUGHTER Malignant catarrhal fever

FARM OF ORIGIN Cleughead, Lesmahagow, Lanarkshire

HOME BRED OR BOUGHT IN Bought in at 4 years of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Occasional on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown

EPIDEMIOLOGY CASE NUMBER: E334

BREED: Hereford X AGE: 7 yrs SEX: Female DATE OF REFERENCE: 28.5.79

CAUSE OF DEATH/SLAUGHTER: Chronic suppurative pneumonia

FARM OF ORIGIN South Mains, Sanquhar, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Rare

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E335

BREED: Aberdeen Angus X AGE: 7 years SEX: Female DATE OF REFERENCE: 28.5.79

CAUSE OF DEATH/SLAUGHTER Intussusception

FARM OF ORIGIN Mains, Sanquhar, Dumfriesshire

HOME BRED OR BOUGHT IN Bought in at 3 years of age

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E336

BREED: Friesian AGE: 8 yrs SEX: Female DATE OF REFERENCE: 28.5.79

CAUSE OF DEATH/SLAUGHTER: Mediastinitis

FARM OF ORIGIN Mid Drumloch, Strathaven, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Rare, only as a heifer

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E337

BREED: Friesian AGE: 8 yrs SEX: Female DATE OF REFERENCE: 29.5.79

CAUSE OF DEATH/SLAUGHTER Endocarditis

FARM OF ORIGIN Bencloich, Lennoxtown, Dumbartonshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E338

BREED: Hereford X AGE: 9yrs SEX: Female DATE OF REFERENCE: 31.5.79

CAUSE OF DEATH/SLAUGHTER: Traumatic reticulitis

FARM OF ORIGIN Burton, Doonfoot, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 1½ years of age

BRACKEN INFESTATION Light (3% of pastures)

ACCESS Frequent when on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E339

BREED: Friesian AGE: 8 yrs SEX: Female DATE OF REFERENCE: 1.6.79

CAUSE OF DEATH/SLAUGHTER Osteomyelitis of ribs

FARM OF ORIGIN Waterbutts, Errol, Angus

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E340

BREED: Ayrshire AGE: 4 yrs SEX: Female DATE OF REFERENCE: 9.10.79

CAUSE OF DEATH/SLAUGHTER: Johnes disease

FARM OF ORIGIN Knockdon, Straiton, Ayrshire

HOME BRED OR BOUGHT IN Bought in at 2½ years of age

BRACKEN INFESTATION Moderate (15% of pastures)

ACCESS Never on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS Unknown. Purchased at market

EPIDEMIOLOGY CASE NUMBER: E341

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 28.8.79

CAUSE OF DEATH/SLAUGHTER Endocarditis

FARM OF ORIGIN Dalvey, Cromdale, Nairn

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Very rare, only as a calf

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E342

BREED: Ayrshire AGE: 5 yrs SEX: Female DATE OF REFERENCE: 15.10.79

CAUSE OF DEATH/SLAUGHTER: Hepatic cirrhosis

FARM OF ORIGIN Kilblaan, Southend, Argyll

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E343

BREED: Friesian AGE: 4 yrs SEX: Female DATE OF REFERENCE: 23.10.79

CAUSE OF DEATH/SLAUGHTER Traumatic pericarditis

FARM OF ORIGIN Auchengree, Stepps, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E344

BREED: Aberdeen Angus X AGE: 6 yrs SEX: Female DATE OF REFERENCE: 6.11.79

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis

FARM OF ORIGIN: Stenhouse Est., Thornhill, Dumfriesshire

HOME BRED OR BOUGHT IN: Bought in at 2 years of age

BRACKEN INFESTATION: Moderate (6% of pastures)

ACCESS: Rare on farm of origin

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: Unknown. Ireland

EPIDEMIOLOGY CASE NUMBER: E345

BREED: Friesian AGE: 5 yrs SEX: Female DATE OF REFERENCE: 16.11.79

CAUSE OF DEATH/SLAUGHTER: Left displacement of abomasum, chronic septic arthritis

FARM OF ORIGIN: Hemphill, Moscow, Ayrshire

HOME BRED OR BOUGHT IN: Homebred

BRACKEN INFESTATION: Light (< 1% of pastures)

ACCESS: Rare

INCIDENTS OF ACUTE BRACKEN POISONING: None

PREVIOUS ORIGINS: -

EPIDEMIOLOGY CASE NUMBER: E346

BREED: Ayrshire AGE: 8 yrs SEX: Female DATE OF REFERENCE: 27.11.79

CAUSE OF DEATH/SLAUGHTER: Endocarditis

FARM OF ORIGIN Monkland, Kilmarnock, Ayrshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E347

BREED: Friesian AGE: 6 yrs SEX: Female DATE OF REFERENCE: 30.11.79

CAUSE OF DEATH/SLAUGHTER Metritis and renal infarction

FARM OF ORIGIN Huntleyhill, Lanark, Lanarkshire

HOME BRED OR BOUGHT IN Homebred

BRACKEN INFESTATION Light (< 1% of pastures)

ACCESS Rare, only as a heiver between 15 and 21 months of age

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E348

BREED: Galloway AGE: 9 yrs SEX: Female DATE OF REFERENCE: 7.12.79

CAUSE OF DEATH/SLAUGHTER: Pyelonephritis

FARM OF ORIGIN Hartside, Lamington, Lanarkshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Severe (> 20% of pastures)

ACCESS Frequent until 6 years of age, never thereafter

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

EPIDEMIOLOGY CASE NUMBER: E349

BREED: Friesian AGE: 5 yrs SEX: Female DATE OF REFERENCE: 11.1.80

CAUSE OF DEATH/SLAUGHTER Traumatic pericarditis

FARM OF ORIGIN Braidfield, Hardgate, Dunbartonshire

HOME BRED OR BOUGHT IN Home bred

BRACKEN INFESTATION Nil

ACCESS Never

INCIDENTS OF ACUTE BRACKEN POISONING None

PREVIOUS ORIGINS -

APPENDIX 4.8

THE REFERRAL FARMS OF IMMATURE ANIMALS WITH MALIGNANT NEOPLASMS.

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E350	08.10.71	Friesian	10 mths	F	Glenwhask,Barrhill,Ayrshire	Multicentric lymphosarcoma
E351	11.10.71	Friesian	2 yrs	F	Dunlop,Mauchline,Ayrshire	Thymic lymphosarcoma
E352	29.10.71	Hereford X	10 mths	F	Beckhall,Canonbie,Dumfriesshire	Thymic lymphosarcoma
E353	25.11.71	Ayrshire	15 mths	F	Holmes Farm,Drybridge,Kilmarnock, Ayrshire	Thymic lymphosarcoma
E354	21.12.71	Aberdeen Angus X	8 mths	F	Fauld of Wheat, New Galloway, Kirkcudbright	Multicentric lymphosarcoma
E355	08.01.72	Friesian	8 mths	M	Thornyhills, Lanark, Lanarkshire	Multicentric lymphosarcoma
E356	17.03.72	Aberdeen Angus	3 mths	M	Kirknan, Glassary, Argyll	Multicentric lymphosarcoma
E357	21.07.72	Galloway X	5 mths	F	Corsebank,Sanquhar, Dumfriesshire	Multicentric lymphosarcoma
E358	24.07.72	Hereford X	2 days	M	Glenbuck,Muirkirk,Ayrshire	Haemangiosarcoma of skin
E359	20.10.72	Shorthorn	8 mths	M	Balgray, Inchture, Perthshire	Thymic lymphosarcoma
E360	26.10.72	Hereford X	12 mths	F	Cogarth Farm,Parton,Castle Douglas,Kirkcudbright	Thymic lymphosarcoma
E361	05.01.73	Jersey	8 mths	F	Muirhall, Hamilton, Lanarkshire	Thymic lymphosarcoma
E362	17.03.73	Hereford X	18 mths	M	Ward of Cairnlea, Barrhill, Ayrshire	Thymic lymphosarcoma
E363	10.05.73	Friesian	12 mths	M	Faulds, West Kilbride , Ayrshire	Thymic lymphosarcoma
E364	23.05.73	Friesian	6 mths	F	Wester Lochdrum, Bonnybridge, Stirlingshire	Thymic lymphosarcoma
E365	05.06.73	Shorthorn X	10 mths	F	Burniehall, Carluke, Lanarkshire	Thymic lymphosarcoma

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E366	07.06.73	Ayrshire	4 mths	F	Burnockstang,Ochitree,Ayrshire	Multicentric lymphosarcoma
E367	07.06.73	Friesian	21 mths	F	Watson, Cumnock, Ayrshire	Thymic lymphosarcoma
E368	12.08.73	Ayrshire	8 mths	F	Davidston, Stair, Ayrshire	Thymic lymphosarcoma
E369	06.11.73	Ayrshire	12 mths	F	Floors, Eaglesham, Renfrewshire	Thymic lymphosarcoma
E370	13.11.73	Galloway	12 mths	M	Loch Mailing, Auldgirth, Dumfriesshire	Multicentric lymphosarcoma
E371	24.11.73	Hereford	12 mths	M	Westfields, Bonchester Bridge, Roxburgh	Multicentric lymphosarcoma
E372	31.12.73	Friesian	18 mths	F	Knockroon, Crosshill, Ayrshire	Undifferentiated sarcoma of abdomen
E373	22.01.74	Shorthorn	3 wks	M	Coulighailtro, Kilberry, Argyll	Multicentric lymphosarcoma
E374	29.01.74	Hereford X	2 wks	F	Drum, Kilfinan, Argyll	Multicentric lymphosarcoma
E375	30.01.74	Friesian	18 mths	F	Shotlynn, Hamilton, Lanarkshire	Multicentric lymphosarcoma
E376	19.02.74	Friesian	2 yrs	F	Altonhill, Kilmarnock, Ayrshire	Granulosa Cell Tumour
E377	10.05.74	Hereford X	15 mths	M	Strathnafanaig, Clachan, Argyll	Skin lymphosarcoma
E378	14.05.74	Friesian	4 mths	F	Barskimming, Mauchline, Ayrshire	Multicentric lymphosarcoma
E379	25.06.74	Hereford X	8 mths	F	Fassfern, Kinlocheil, Inverness-shire	Thymic lymphosarcoma
E380	08.10.74	Friesian	6 mths	F	Broadlea, Eaglesfield, Dumfriesshire	Thymic lymphosarcoma
E381	16.10.74	Friesian	6 mths	F	Hall of Carnduff, Strathaven, Lanarkshire	Thymic lymphosarcoma

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E382	30.01.75	Aberdeen Angus	12 mths	F	Geddes Holm Farm, Nairn, Nairn	Thymic lymphosarcoma
E383	11.02.75	Friesian	18 mths	F	Ditton, Symington, Ayrshire	Thymic lymphosarcoma
E384	07.05.75	Hereford X	15 mths	F	Bogside, Langbank, Renfrewshire	Thymic lymphosarcoma
E385	03.06.75	Aberdeen Angux X	3 mths	F	West Brown Castle, Strathaven, Lanarkshire	Multicentric lymphosarcoma
E386	10.06.75	Friesian	21 mths	F	Shutterflat, Beith, Ayrshire	Thymic lymphosarcoma
E387	23.08.75	Aberdeen Angus	21 mths	F	Simperim, Meigle, Perthshire	Multicentric lymphosarcoma
E388	04.12.75	Friesian	2 yrs	M	Deanfoot, West Linton, Peebles	Skin lymphosarcoma
E389	17.02.76	Friesian	2 yrs	F	Wester Auchencarroch, Jamestown Dumbartonshire	Multicentric lymphosarcoma
E390	04.05.76	Shorthorn X	12 mths	F	Riddleton Hill, St. Boswells, Roxburgh	Thymic lymphosarcoma
E391	07.05.76	Hereford X	14 mths	M	Friarshall, Melrose, Roxburgh	Thymic lymphosarcoma
E392	21.05.76	Aberdeen Angus X	12 mths	F	Kintyre, Kirriemuir , Angus	Thymic lymphosarcoma
E393	18.06.76	Friesian	4 mths	F	Knowetap, Sandford, Lanarkshire	Multicentric lymphosarcoma
E394	03.06.76	Aberdeen Angus X	6 mths	M	Bogg, Sanquhar, Dumfriesshire	Multicentric lymphosarcoma
E395	15.09.76	Galloway	4 mths	F	Loch Mailing, Auldgirth, Dumfriesshire	Thymic lymphosarcoma
E396	21.10.76	Charolais X	2 wks	M	Cocklaw, Ayton, Berwickshire	Multicentric lymphosarcoma

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E397	21.10.76	Galloway X	2 wks	F	Balvicar, Argyll	Multicentric lymphosarcoma
E398	25.11.76	Hereford	3 mths	F	Over Hazelfield, Auchencairn, Kirkcudbright	Multicentric lymphosarcoma
E399	10.12.76	Ayrshire	10 mths	M	Bridgend Mains, Stair, Ayrshire	Thymic lymphosarcoma
E400	31.12.76	Ayrshire X	18 mths	F	Barrhill, Auldgirth, Dumfriesshire	Multicentric lymphosarcoma
E401	08.07.77	Charolais	12 mths	F	Milton, Kirkcudbright, Kirkcudbright	Thymic lymphosarcoma
E402	25.02.77	Hereford	4 mths	F	High Barcaple, Ringford, Kirkcudbright	Thymic lymphosarcoma
E403	25.02.77	Friesian	2 yrs	F	Turningshaws, Bridge of Weir, Renfrewshire	Multicentric lymphosarcoma
E404	26.03.77	Aberdeen Angus	8 mths	F	Tillyewe, Udwy, Aberdeenshire	Thymic lymphosarcoma
E405	27.04.77	Hereford X	15 mths	F	Barrodger, Lockwinnoch, Renfrewshire	Undifferentiated sarcoma of cranial cavity
E406	09.06.77	Aberdeen Angus X	6 wks	F	Peterseat, Nigg,Aberdeenshire	Multicentric lymphosarcoma
E407	20.06.77	Hereford X	5 mths	F	Birfwick, Closeburn, Dunfriesshire	Multicentric lymphosarcoma
E408	20.07.77	Hereford X	4 mths	M	Carlinside, Lanark, Lanarkshire	Multicentric lymphosarcoma
E409	15.08.77	Ayrshire	2 wks	F	Garfield, Mauchline, Ayrshire	Multicentric lymphosarcoma

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E410	25.11.77	Ayrshire	2 yrs	F	Glenreasdell, Whitehouse, Argyll	Thymic lymphosarcoma
E411	14.01.78	Hereford X	4½ mths	F	Watherstone, Stow, Mid Lothian	Multicentric lymphosarcoma
E412	22.01.78	Hereford X	10 mths	M	Linburn, Bishopton, Renfrewshire	Multicentric lymphosarcoma
E413	05.06.78	Hereford X	15 mths	M	Quixwood, Duns, Berwickshire	Skin lymphosarcoma
E414	27.06.78	Hereford X	18 mths	F	Shieldhill, Newton Mearns, Renfrewshire	Multicentric lymphosarcoma
E415	04.11.78	Ayrshire	8 mths	M	Windylodge, Dalry, Ayrshire	Malignant Thyroid Tumour
E416	11.11.78	Hereford X	5 mths	F	Barr, Newton Mearns, Renfrewshire	Multicentric lymphosarcoma
E417	16.11.78	Friesian	7 mths	F	Woodhouse, Mayfield,Bootle, Lancashire	Multicentric lymphosarcoma
E418	29.11.78	Friesian	15 mths	F	Hatton, Bishopton, Renfrewshire	Malignant Ovarian Teratoma
E419	28.12.78	Hereford X	5 mths	M	South Houret Dalry, Ayrshire.	Multicentric lymphosarcoma
E420	10.0.79	Friesian	3 mths	M	Nether Fingland, Elvanfoot, Dumfriesshire	Maligant Teratoma
E421	21.02.79	Friesian	8 mths	F	Bonanhill, Strathaven, Lanarkshire	Thymic lymphosarcoma
E422	21.02.79	Friesian X	4 mths	F	Greenhill, Cleland, Lanarkshire	Multicentric lymphosarcoma

Case No.	Date of Admission	Breed	Age	Sex	Source	Neoplasm
E 423	20.04.79	Friesian	4 mths	M	Fairfield, Kippen, Stirlingshire	Mesothelioma
E 424	22.04.79	Aberdeen Angus X	7 mths	M	Blackford, Croy, Invernesshire	Thymic lymphosarcoma
E 425	03.05.79	Ayrshire	18 mths	F	Shacklehill, Mossblown, Ayrshire	Thymic lymphosarcoma
E 426	27.07.79	Ayrshire	6 mths	F	Langstilly, Lochwinnoch, Renfrewshire	Multicentric lymphosarcoma

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